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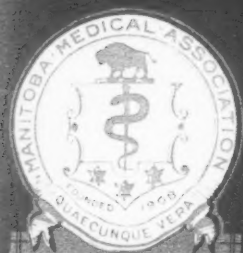
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The Manitoba Medical Review

Vol. 35

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Medicine

Clinical and Therapeutic Aspects of Sprue

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Before considering the general clinical aspects of sprue it seems well to relate it to those conditions with which it might be confused by virtue of the presence of steatorrhea. Recent workers seem to agree that the basic defect in sprue is a defect in the absorption of fat, fat soluble vitamins and certain carbohydrates with secondary malabsorption of minerals. A convenient classification of the malabsorption syndrome and one that appears to be easily applicable to clinical states is that used by Cross in a recent discussion of this state.³⁰ The particular syndrome which interests us today is that under group 3 in this classification.

Classification of the Steatorrheas.

1. Defects in digestion and/or emulsification.
 - a. Hepatic—obstructive jaundice, hepatitis.
 - b. Pancreatic — cystic fibrosis, chronic pancreatitis, after pancreatectomy.
 - c. Gastric—after gastrectomy.
2. Lack of Small Intestine.
 - a. After resection.
 - b. Fistulae.
3. Defects in absorption—mechanism obscure.
 - a. Coeliac disease.
 - b. Sprue (believed to be same disease process).
4. Disease involving intestinal wall.
 - a. Regional enteritis.
 - b. Scleroderma.
 - c. Amyloidosis.
5. Diseases of Intestinal Lymphatics.
 - a. Tuberculosis.
 - b. Hodgkin's Disease.
 - c. Intestinal Lipodystrophy.
6. Rapid Transit.

Moderate steatorrhea may be associated with diarrhea of any cause.

Nomenclature.

It is necessary at this point to clarify the nomenclature as it will be used in this paper and also to discuss some of the reasons why coeliac disease, tropical sprue, non-tropical sprue and idiopathic steatorrhea are considered by some observers to be only geographic and age variations in the same disease process. Many workers prefer to discard the use of the terms "tropical" and "non-tropical" and to include all cases of this

syndrome in adults under the term "idiopathic steatorrhea" while those cases in children are referred to as "celiac disease" although it is admitted that this name is neither accurate as to clinical description, nor as to etiology. However, in the absence of a more accurate knowledge regarding etiology and because of common usage, the name can properly be retained for the present. That the identity of the disease process was recognized as being similar by Gee has been mentioned previously and this notion was also advocated in Thaysen's classic monograph in 1932³⁷ and also by most other serious students of the subject^{27, 29, 30} St. Agnese refers to the diseases as adult and childhood coeliac disease.³⁰ While, as mentioned, the etiology is obscure, evidence has been accumulating that there is an hereditary constitutional defect in metabolism which persists after the clinical manifestations of the severe illness have disappeared. The defect is transmitted as a dominant trait of variable penetrance and expressivity. This defect may manifest itself at different ages, either because of inherent differences in the severity of the disease itself or because of the occurrence of appropriate extrinsic conditions. It may also remain mild or latent for an indefinite period of time and manifest itself at times later in life when appropriate trigger mechanisms are set off. Trigger mechanisms are varied and may consist of acute enteral or parenteral infections, dietary deficiencies, administration of a wide spectrum antibiotic⁵² or often psychic trauma. The same defect may lead to different but comparable disease pictures at different ages and under different conditions.

Although it was originally believed that sprue only occurred in tropical countries, it gradually became clear that a similar disease was being frequently seen in more temperate climates. Originally it was felt that it was still necessary to have had residence in the tropics at some time or other but this idea was gradually relinquished when reports increased of a typical sprue picture in patients, who had never left temperate zones. Although many workers still insist on distinguishing between "tropical" and "non-tropical" sprue, chiefly on the basis of disturbed calcium metabolism with skeletal deformities in the latter, the general consensus, at least in America and Scandinavia favors the one basic defect with geographic variants (or perhaps racial variants).

Snell discusses the problem of the interrelationship of the various sprue syndromes in 14 cases of sprue contracted while in the tropics with 32 cases of non-tropical sprue seen at the Mayo Clinic.²⁹

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Both diseases appear to be essentially the same both clinically and pathologically with the exception of the more seriously disturbed calcium metabolism in non-tropical sprue. This of course leads to skeletal deformities and infantilism. Autopsy studies failed to reveal any significant differences between tropical and non-tropical sprue and if any changes were present they did not clearly differentiate one from the other. In those cases showing skeletal and growth changes one usually finds an earlier onset or a history suggestive of celiac disease in childhood and it would then be expected that the disease might have different manifestations, particularly as regards the effect on the skeletal system. Changes in the skeleton are said to be almost unknown in tropical sprue. The greater exposure to the sun may have a bearing in this respect. In other respects, vitamin deficiencies, plasma protein levels and subacute combined degeneration are more common in non-tropical sprue and many assume that tropical sprue is a milder form of the disease. This may be partially true but the possibility also exists that the diagnosis is made much earlier in the tropics where physicians are alerted to the diagnosis. In temperate climates the diagnosis is often delayed for years and then changes are often severe and irreversible. Snell feels that if we become more conscious of the disease then chances are that the distinctions between tropical and non-tropical sprue will become less and less. Certainly, Andersen and di Sant'Agnese, who are perhaps the most serious students of the disease in this country, speak only of adult and childhood celiac disease. They relate the differences in clinical picture only to the irreversible changes which take place due to age and long standing disease.

The Incidence and Genetics of the Sprue Syndrome

Definite figures as to the incidence of the sprue syndrome are limited even in those countries with a high incidence. The advent of the National Health Scheme in Britain made it possible to collect figures of the incidence of this disease because of the necessity to apply for special rations. These figures, although helpful, must be interpreted with caution since it was found that the annual incidence rose or fell, to some degree, depending on government policy as to strictness with which special diets would be allowed. However, the figures have a certain value and have been reported by Davidson and Fountain⁹. In a six year period there were 2,044 cases reported as follows:

Celiac disease	1303 cases
Non-tropical sprue	311 cases
Tropical sprue	403 cases
Unclassified	27 cases

The average annual incidence in the British Isles was calculated to be 117 cases or 1 case per 135,000 population for celiac disease and 44 cases or 1 case per 700,000 population over 15 years of

age. The sex incidence was about equally divided. An interesting aspect of this study of incidence was the occurrence of the syndrome in families. Questionnaires were sent to 100 family doctors in charge of cases of non-tropical sprue and control questionnaires were sent to 100 normal people. 34% of the patients with non-tropical sprue were either proved cases of celiac disease in childhood or had a history of alimentary disorders in childhood, while in only three of the controls was there any indication of malabsorption or intestinal disease. 200 cases of the celiac disease were also investigated in this way and it was found that 9.2% of them gave a family history of celiac disease in siblings and 3.8% gave a family history of sprue.

Bartley³⁹ reports steatorrhea in a father and four children but on reading his protocol one cannot be sure that these represent sprue syndrome rather than fibrocystic disease of the pancreas.

Traditional descriptions refer only to the advanced cases of the disease. In the severe form it is not very common but in mild form it is widespread in children and perhaps in adults³⁰. The sprue syndrome occurs in the white race regardless of national origin. It seems to be uncommon among negroes and has apparently never been reported among Orientals³⁰. The familial occurrence of celiac disease, stressed first by Andersen, is a strong argument in favor of the existence of a basic hereditary constitutional defect. According to this concept, enteral or parenteral infections which, especially in children, commonly initiate the manifest disease, act as trigger mechanisms precipitating a symptom-complex which might otherwise have remained latent or unrecognized. They therefore play a secondary and not a primary role in the etiology of the sprue syndrome. Celiac disease and the sprue syndrome are most frequently encountered in subjects below the age of five years or above the age of twenty-five years. If one assumes a linkage between these two syndromes it should be interesting to know what happens to children with celiac disease after they have gone beyond the usual age of celiac disease. di Sant'Agnese studied 23 children who had earlier been studied because of celiac disease by Andersen. The second study took place 5 to 13 years later and although all these patients were clinically well many of the chemical tests of intestinal absorption and stool fat content were found to be grossly abnormal. These workers concluded that this stage represented a latent period in the disease and that these patients might well expect further trouble later in life. Since a followup of these patients would require another thirty or forty years observation, a further group of patients with sprue were studied as to past history of G.I. disturbance or celiac disease. Ten adult patients were thus studied. All had proven sprue. In six of the ten patients there was either a family incidence of celiac disease or history of the onset of the disease

in childhood. In two of the patients both of these findings were present.

The Clinical Picture of the Sprue Syndrome

Many excellent studies have been made on all aspects of the sprue syndrome, both collectively and separately as studies of tropical, non-tropical sprue and as celiac disease. Since our interest is chiefly in the adult form of the disease, what follows will be concerned only with the disease as manifested in older patients. In essence, of course, the clinical picture is agreed upon by all observers, so long as one takes into account the divergence of opinion, in various countries as to whether tropical and non-tropical sprue are the same process with geographic variation. Most of the observations which follow are a summation of the reports of Hanes²⁷, Adlerberg and Schein³⁹, Bennett et al¹, Stefanini⁴³, Snell²⁹, Cooke et al⁷, Castle et al¹² and others.

General Observations

Practically all cases of sprue are characterized by lassitude, loss of weight and a long history. Cooke stresses the persistence of lassitude even after diarrhea had abated and anemia had been corrected. He suggests that this symptom may have an organic basis, especially since neurotic symptoms were remarkable for their rarity. In spite of great physical weariness, the mental activity and initiative of these patients compared favorably with the normal. Lassitude and loss of weight occurred in 97% of Cooke's series. In all series the ratio between males and females was very close to 1:1.

One of the characteristic features of sprue, and particularly when it occurs in temperate climates is its chronicity. Adlersberg et al in a series of 36 cases of sprue found duration of symptoms prior to hospitalization to be 80 months; Thaysen^{10, 11} found 50% of his cases gave a history extending back in childhood and Cooke⁷ had a similar experience when 43 patients in a series of 100 cases also dated the onset of symptoms into infancy or childhood; Snell's series²⁹ also showed that a majority of the patients were seen three or more years after the onset of the earliest symptoms; Bennett reported 11 of 15 cases studied gave a history dating from infancy, and in three others significant symptoms were present before the age of 13 years. In only one instance in this series was there a negative history until the age of 52.

These figures appear to show that failure to interrogate about infancy or childhood symptoms may be the main reason for so many so-called "spontaneous" cases. It is reasonable to conclude that steatorrhea associated with tetany, anemia and osteomalacia is a disease originating in childhood, though not necessarily recognized until adolescence or adult life is reached. It is only fair to state that in cases of sprue occurring in the tropics the period of time elapsing between earliest symptom and hospitalization is much shorter.

Stefanini⁴³ gives no average time but infers intervals of only a few weeks.

Loss of weight is an outstanding feature in sprue and may be alarming in extent. It occurred in 97% of Cooke's series and Hanes states that it is practically a constant finding and may equal or exceed that found in any other condition. Losses from 25 to 70 pounds are the rule and not infrequently the weight may be halved. Thaysen^{10, 11} noted an average loss of weight of 18Kg (39.5 lbs.) and Snell²⁹ an average loss of 30 lbs., the greatest being 70 lbs. The highest mortality appears to occur in those with the greatest weight loss.

Gastro-intestinal Disturbances^{1, 7, 10, 11, 27, 29, 30, 36}

Included in this group are flatulence, pain or abdominal discomfort, glossitis, nausea and vomiting, diarrhea and steatorrhea and, of course, these symptoms are often outstanding in the sprue syndrome.

Glossitis is more common in tropical sprue than in non-tropical sprue and appears to vary from 33% in the latter to 90% in the former. Abdominal distension may be conspicuous in some cases but is not present in every case. Cooke⁷ reports a 50% incidence with abdominal discomfort appearing in 31%. Diarrhoea is very frequent but not invariable in sprue and, in fact, there may be episodes of constipation. In spite of the frequent absence of diarrhea, however, steatorrhea is an invariable finding and, of course, the diagnosis cannot be made in the absence of steatorrhea. Gastro-intestinal symptoms are frequently the presenting symptoms in this syndrome. Diarrhea may be intermittent at first and perhaps more constant as the disease progresses. The stools are usually paler than normal, mostly offensive and sometimes frothy. The onset of episodes of diarrhea may be sudden and aggravated by fatigue, exertion, emotional upheaval or intercurrent infections and by over-indulgence in fatty foods. Between attacks more than 70% of patients had normal bowel habits and passed stools of normal color and consistency. It should also be emphasized that pale stools may occur in the absence of steatorrhea and that frequently stools containing excess fat may be of normal color and consistency.

Abnormalities of the Skeletal System

Bone deformities were reported in 7 of 15 cases by Bennett et al¹ but in none of the 15 cases was there a normal skeleton. The commonest deformities were dwarfism, genu valgum, genu varum, bent or bowed bones, beaded ribs, pathological fractures, splayed rib margin, distorted pelvis and flaring of the wrists. Rickets was very common if the disease was of long standing and both x-ray and chemical evidence of rickets may be manifest. Rickets is most likely to occur during periods of active growth but if growth is prevented by the severity of the disease then only osteomalacia may be present. Osteoporosis was present in 12 of 15 cases reported by Bennett. These

changes are common in non-tropical sprue but uncommon in tropical sprue and, indeed, Stefanini does not discuss them in his monograph on tropical sprue⁴³. He records calcium and phosphorus levels which appear slightly lower than normal but not so much so in cases of non-tropical sprue. This fact has been used as the basis for differentiation of some cases but it is thought improper to do so by most writers.

Muscle cramps are common in sprue but are not necessarily related to the level of calcium or phosphorus. Tetany is reported in a variable number of cases by different authors and may vary from 2% to 25%. If frank tetany does not occur there may be a positive Chovstek's or Trousseau's sign.

Change in the Skin

Thaysen noted pigmentation in 12 of his 34 cases¹⁰ and Bennett et al report an incidence of skin lesions in 7 of 15 cases. The lesions may consist of scattered diffuse brown patches or they may resemble pellagrinous changes in the skin. Snell²⁹ emphasized the frequent confusion between pellagra and sprue and reported that only 10 of his 32 cases showed no pigmentation. Petechiae may occur. Brittle nails and clubbed fingers may occur. The pigmentation may frequently have the same distribution as chloasma.

Miscellaneous Findings in Sprue

Fever has been reported to occur in non-tropical sprue and is usually low grade. If a high fever occurs some other basis than sprue should be sought for⁷. Stefanini reports fever in tropical sprue and states that those cases with fever usually had a much more serious prognosis⁴³.

Edema and ascites may occur when the general condition is poor and hypoproteinemia occurs. Edema and/or ascites were present in 34% of Cooke's cases⁷.

Snell²⁹ reported hypotension as a common finding in his series. He reported a B.P. above 110 systolic in only 5 of 22 cases. Cooke has a similar experience and Stefanini⁴³ reports an incidence of hypotension of 92.4% in tropical sprue. In Stefanini's series the average systolic B.P. was 108 mm. Hg. and diastolic average was 64 mm. Hg.

About 1-2% of cases of sprue will present the changes of a subacute combined degeneration of the spinal cord.

The pulmonary system is not usually involved but if infection is present one should be doubly sure he is not dealing with a pancreatic steatorrhea.

Laboratory Aids in the Diagnosis of Sprue

The laboratory diagnosis of the sprue syndrome is exceedingly important and can conveniently be described under four headings:

- 1) Blood Studies
- 2) Tolerance Studies
- 3) Stool Studies
- 4) X-ray Studies

Blood Studies.

Anemia is another of the classic findings in sprue and, indeed, it is felt by some to be a necessary feature before diagnosis can be made. Cooke found it in 100% of his cases⁷ and in this group 66% had a macrocytic anemia, often indistinguishable from pernicious anemia, while 34% had a hypochromic anemia. Bennett et al¹ and Aldersberg et al³⁹, Snell²⁹, and Hanes²⁷, and Castle et al^{2, 3, 4} report anemia in 100% of their cases. All writers agree that approximately 50 - 66% of cases will show a macrocytic anemia, a further 33 - 50% will show a hypochromic anemia. The anemia of coeliac disease is almost invariably a hypochromic microcytic anemia, since children rarely respond with a macrocytosis to any stimulation.

Bone marrow studies³⁷ confirm the peripheral blood studies and show a megaloblastic arrest in 50% of cases, a normoblastic marrow in 30% and the remaining 20% had a marrow which had characteristics of the megaloblastic marrow, but there was some maturation to the normoblastic series. The marrow in coeliac disease is usually normoblastic. If the patient is cachectic and near death, the marrow may be fatty.

Adlersberg and Schein³⁹ found that if the hemoglobin was below 5 grams, bleeding was usually a prominent symptom and may have been a reflection of the hypoprothrombinemia.

Calcium and phosphorus levels in the blood have been commented on earlier. The alkaline phosphatase is not affected unless there are active bone lesions, and then it is elevated.

Cooke⁷, Lubran et al⁴⁰ and Cooke⁴⁹ report the incidence of hypokalemia, especially in the active phase of the disease with diarrhea. Lubran states that there is a negative potassium balance during diarrhea which remains untreated and that the symptoms and EKG changes of hypokalemia may occur and may be reversed by treatment of the sprue. Cooke states that hypokalemia occurs in approximately 20% of patients with sprue and he believes it may be a common cause of death. He feels that 50% of his cases died in that way.⁴⁹ The hypokalemia is not considered to be due to malabsorption, per se, but rather to the increased excretion in the feces.

Tolerance Studies.

Since sprue is considered to be essentially the result of defective gastrointestinal absorption, it seems reasonable that certain tests of the absorptive capacity of the intestinal lumen would be perfected as an aid in the diagnosis of the syndrome. These tolerance tests may be done utilizing carbohydrates, fats and fat soluble vitamins or water.

Glucose Tolerance Tests.

The oral glucose tolerance curve in cases of sprue is a flat curve and this finding has been

confirmed by all investigators. Hanes²⁷ states that in order for the curve to be considered a flat curve, there must be a rise of less than 40 mgm/100 cc. above the fasting glucose level on a dose of 1 gram of glucose per kilo of body weight. In 54 of his cases, the average rise was 20 mgm/100 cc. Hanes' criteria appear to have been pretty well accepted by most observers. The intravenous glucose tolerance test is, of course, within normal limits.

Fat Tolerance Tests.

Tests to evaluate the defect in fat absorption in sprue have taken two forms: a) The vitamin A tolerance test and b) fat excretion studies.

Like the glucose tolerance curve, the vitamin A tolerance curve is flat in sprue. Serum carotene levels are abnormally low in any condition which leads to steatorrhea³⁰, but fasting vitamin A levels may be normal because they reflect the preformed vitamin A which has been fed in the few days prior to the test. However, if a standard dose of vitamin A is fed (7000 units/Kg. orally) and the blood levels studied subsequently, it is found that the curve remains flat and suggests malabsorption. This applies to all the fat soluble vitamins, A, D, E and K and the lack of the latter is responsible for much of the hypoprothrombinemia and hemorrhagic tendencies to be found in this syndrome.

Fat excretion studies are most often done as measurements of excreted fat related to fat intake for a given period of time. Most observers agree that sprue cannot be diagnosed positively without a knowledge of the fat excretion data. Studies are made both of the total amount of fat excreted as well as the type of fat. In normal subjects, variations in fat intake within reasonable limits do not appreciably change fecal fat, but in steatorrhea, an increase in dietary fat proportionately increases fecal fat³⁶. Fourman et al³⁶ in a series of 12 days' balance studies, found the mean daily fat excretion in normals to be 2.88 grams while in cases of sprue the mean fat excretion was 59 grams. All patients were on diets containing 70 grams of fat and 70 grams of protein. At the same time, the fecal nitrogen loss is low as contrasted to pancreatic steatorrhea in which both fat loss and nitrogen loss is great.

In Hanes' series²⁷, the average fecal fat was 48.5% and Snell²⁹ found fecal fat usually over 30% of that ingested, calculated from dry weight of stool. Most of the fecal fat was split or neutral fat. Bennett et al¹ report steatorrhea in all of their cases ranging from 45-71%, divided as follows:

Neutral fat	2.1 to 45.0%
Free fatty acid	9.6 to 28.4%
Combined fatty acid	4.2 to 49.5%

This is in contrast to pancreatic steatorrhea in which the level of free fatty acids is always above 30%, even when the total fecal fat is the same as

in sprue. Much of the fecal fat in sprue is excreted as soaps combined with calcium. The high fecal calcium is considered to be the main cause for the low serum calcium. However, calcium loss does not appear to be directly related to the fecal fat content. In fact, there is a very crude relationship.³⁰ Another and more likely explanation is that vitamin D is fat soluble and hence, because of fecal loss, not available to aid in the absorption of calcium. However, calcium loss is not directly related to vitamin D lack and so many factors may play a part or there may be a specific element of calcium malabsorption, just as there is for glucose or fats.

Water Tolerance Tests.

Recently Wollaeger et al³⁴ and Taylor³³ have reported on the delayed diuresis in patients with sprue following a given water load. Wollaeger et al noticed in 7 patients with nontropical sprue, that under standard conditions of food and water intake, all patients were found to have an abnormally large volume of urine during the night. Their studies suggested that the nocturnal diuresis was related to the retention of large volumes of water in the small intestine during an abnormally prolonged period of digestion and absorption of food. When absorption was finally completed, the retained water required by this process was excreted, causing the nocturnal diuresis.

Roentgen Studies in Sprue

The roentgen picture of the small bowel in sprue is sufficiently characteristic to be of diagnostic significance in 70% of the patients.⁵ Although there is nothing pathognomonic of sprue on x-ray study, there are several very suggestive changes which may be described as follows:

1. Dilatation—this is one of the most constant findings and usually best visualized in the mid- and distal jejunum. In general, dilatation in Adlerberg's series⁵ appeared related to the severity of the disease and was present in 37 of 40 cases studied and varied from 1.5 times normal to 3 times normal (normal 2-3 cm.). The small bowel may be dilated as well and this is believed to be related to the increased incidence of volvulus in patients with sprue. Dilatation is an important finding because the "deficiency pattern" as it occurs in other deficiency states usually does not show the marked dilatation that is present in sprue.

2. Segmentation occurred in 34 of 40 reported cases. It was most pronounced in the ileum and best seen in the most severe cases and those associated with hypersecretion. Segmentation is not specific for sprue. The exact etiology of segmentation is unknown, but probably hypersecretion and the presence of large amounts of fat in the small bowel are principle factors.

3. Hypersecretion—an abnormal amount of secretions in the gastro-intestinal tract is a con-

stant roentgen finding in most cases showing the sprue pattern.

4. Thickening of the folds due to edema of the mucosa and hypertonicity of the muscularis mucosae is often seen.

5. Moulage Sign—was a name originally given to the roentgen appearance of segments of the proximal jejunum in sprue in which the folds are smooth and appear to be completely effaced. It appears to be due to marked atony and atrophy of the bowel wall and is found in the most advanced cases.

In differentiating the sprue syndrome from other diseases which cause similar pictures, nothing is absolute from the radiologic point of view, but it is true that: 1) Dilatation, 2) Segmentation and 3) Hypersecretion are much more prominent in sprue than in the other conditions and in that order.

Several observers, principally the British^{59, 60, 61} have stressed the importance of using non-flocculating suspensions of barium to study mucosal pattern in this disease and have shown the relationship of flocculation to the presence of mucus. Many of these patterns could be induced in normal subjects by the use of aqueous suspensions of barium or suspensions containing saline, fat, etc.

X-ray studies of the skeletal system may show osteoporosis, deformities, changes of rickets, etc. and need not be discussed further.

Miscellaneous Laboratory Findings in Sprue.

1. Gastric Analysis—There will be an occasional patient with achlorhydria after histamine, but 79% have been found to have some degree of acidity after histamine.²⁷ This is an important point in the differentiation from P.A.

2. Serum Proteins—tend to be low normal or generally low with a normal A/G ratio. In the terminal stages of the disease or in the acute exacerbations, they may be markedly lowered and edema and ascites may be present on the basis of hypoproteinemia.

3. Prothrombin Time—may be prolonged due to lack of absorption of vitamin K from bowel. Parenteral vitamin K will correct this quickly.

4. Pancreatic Enzymes—have been found to be normal in contrast to low or absent pancreatic enzymes in fibrocystic disease of the pancreas.

5. Blood Cholesterol—often has been found to be reduced in proportion to the diarrhoea.

6. BMR is usually normal although Thaysen reported many elevated BMR's. His findings have not been confirmed in this respect.

The Pathological Picture.

Very little need be said about this aspect of the disease since no changes have been observed in any postmortem studies which are consistent and which appear distinctively a part of the sprue picture. The most characteristic finding, apart

from the extreme wasting, is the lack of findings.³⁹

Criteria for Diagnosis.

A certain amount of confusion arises in the literature when different criteria are used for the diagnosis of the sprue syndrome, and one has the feeling that certain reported cases may not be true cases of sprue. For this reason, the diagnostic criteria, as set down by Hanes²⁷, are enumerated.

1. Steatorrhea—Sprue cannot be diagnosed in the absence of steatorrhea, demonstrated quantitatively, though steatorrhea does, of course, occur in other diseases.

2. Loss of weight—This is a constant finding and may equal or exceed that found in any other condition.

3. Low Glucose Tolerance Curve—a rise of less than 40 mgm/100 cc. after 1 gram glucose/Kg. body weight. If the patient has received liver, folic acid or B12, the rise may exceed 40 mgm/100 cc. since under the influence of therapy, the sugar tolerance curve rises rather rapidly.

4. Anemia—usually macrocytic and hyperchromic, but as stated earlier, may also be normochromic and occasionally hypochromic, especially in children.

5. Hypochlorhydria and Achlorhydria—79% of patients suffering from sprue can be differentiated immediately from P.A. by the presence of HCl after histamine.

To the above criteria, Cooke⁷ adds the finding of the "deficiency pattern" on small bowel x-ray studies.

Therapy of Sprue.

The various modalities in the treatment of sprue will be outlined. The rationale for the use of most of them is easily understood. In spite of intensive therapy, some cases of sprue fail to respond and this appears to be true of nontropical sprue in greater degree than tropical. In general, however, the prognosis is good if treatment is begun early and continued for sufficiently long periods. During treatment, symptomatology may decrease or completely disappear, while the biochemical aberrations may or may not change. This appears to vary from case to case with no known way of forecasting behaviour in any given case.

1. **Diet**—The usual diet prescribed is a high protein, high caloric diet with low fat content and a moderate amount of easily assimilable carbohydrate. During the acute phase, it may be necessary to restrict their carbohydrate, but this appears less likely in adults than in children. Bananas have become a useful food in this circumstance. Avoidance of wheat gluten appears advisable as recent work seems to incriminate it as a trigger mechanism or perhaps as a food causing a more basic disturbance.^{32, 30, 31}

2. **Liver Extract and Related Substances**—Liver extract has rightfully occupied a high place among

the available therapeutic agents for this disease. Treatment with B12 appears to be just as efficacious, although many workers still prefer crude liver extract.^{12,13,35} Folic acid and folinic acid have also been used with great success, especially in tropical sprue.^{28,62,30,35,63}

3. Vitamin and Mineral Therapy—Since there are deficiencies in many other vitamins, a good multivitamin preparation, especially containing B complex, should be prescribed. Vitamin K should be given regularly parenterally. Iron may be given as indicated by the anemia and it may be necessary to give it parenterally.^{30,65} Calcium should be given if serum Ca is low.

Detergents—Emulsifying agents have been reported as useful in sprue by several investigators. Tween 80 1-2 gm. with meals was used by Jones with apparent increase in absorption.

5. A.C.T.H. and Cortisone—These substances have been used with outstanding success, particularly in cases refractory to other therapy by many workers in America and Britain.^{12,16,15,18,66} It seems to be agreed that suppressive doses are required at first; that reduction in dosage should not be too rapid and that a maintenance dose should be maintained for a long time.

6. Blood transfusions are indicated whenever anemia is marked enough to cause debility or where, for some other reason, elevation of blood count is desired fairly rapidly. This does not take the place of specific anti-anemic therapy.

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Surgery

Tumours of the Thymus

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Tumours of the thymus are rare. Since July, 1947, there have been ten patients operated upon in the Winnipeg General Hospital for thymic masses. Enquiries at the Manitoba Cancer Relief and Research Institute added no cases. There are over twenty thousand cancer patients listed there, but without a diagnostic index the information is difficult to extract.

Because of our ignorance of the function of the thymus; the diversity in histological types of tumours; the disparity between histological appearances and subsequent clinical courses; the relationship between myasthenia gravis and the thymus, with and without thymic tumours; we considered it worthwhile to study our experience in this hospital.

Embryology—Convincing evidence has been produced by Norris,¹⁶ who states the thymus is developed from the third branchial complex, both endoderm and ectoderm contributing. He finds the ectodermal sinus is the primordium of the primitive thymic cortex and the source of Hassall's corpuscles. The epithelium of the endodermal pouch gives rise to the syncytial cytotreticulum. The bulk of the gland consists of thymic lymphocytes which have secondarily invaded the gland. At birth, the thymus is relatively large and then fails to keep pace with the general rate of growth. The

normal adult's thymus may range widely in weight, from one or two grams to twenty or more grams, but it is always present.

Physiology—The function of the thymus is not known. Galen believed it suspended and protected the underlying great veins. Glisson, and later Sir Astley Cooper, suggested the thymus had some function in controlling foetal growth. Sir Astley Cooper pointed out the occasional association of thymic enlargement with toxic goitre. Attempts to demonstrate a thymic hormone have been unsuccessful.

Tumours—Lowenhaupt,^{14, 15} has reported detailed studies of the histology of thymic tumours. She has attempted to correlate the tumour appearance with the clinical course. Anterior superior mediastinal masses which had been biopsied, or excised, or obtained at post mortem, were studied. Metastatic tumours and those masses which were part of a generalized lymphoma were discarded. Sixteen cases with unicentric thymic tumours were studied and reported in 1948, and nine additional cases in 1951. The conclusion is that these cases form a closely related series of epithelial derivatives. These could be related to stages in the developing thymus. The grouping is as follows:

Group 1—Carcinoma of the primitive epithelial reticulum, rapidly growing and invasive.

Group 2—variegated-cell carcinoma, with or without the formation of abortive Hassall's corpuscles, likewise a rapidly growing and invasive

Table I

Hospital No.	Patient	Sex	Age at Onset	Presenting Symptom	Time Onset to Surgery	SURGERY		Radiation Therapy
						Apparently Complete	Incomplete	
1	7937/47 9916/47	C.B.	F	36	Pain over left breast	5 mos.	x	Pre-op. x-ray treatment without effect
2	12953/47	M.L.	F	31	Myasthenia Gravis	2 mos.	x	0
3	A4708	M.R.	F	27	Routine chest x-ray	1 mo.		x Post-op. x-ray treatment with temporary relief
4	A19039	H.W.	F	62	Routine chest x-ray	11 mos.	x	Post-op. x-ray treatment
5	A15500	R.S.	M	33	Routine chest x-ray	1 mo.		x Post-op. x-ray treatment
6	A35773	E.V.	M	52	Myasthenia Gravis	6 mos.	x	Post-op. x-ray treatment
7	A43650	E.T.	M	34	Myasthenia Gravis	5 mos.	x	Post-op. x-ray treatment
8	A56797	M.T.	M	36	Myasthenia Gravis	39 mos.		x Post-op. x-ray treatment
9	A75608	A.R.	M	57	Routine chest x-ray	2 mos.	x	0
10	A73845	A.C.	M	56	Superior vena caval syndrome	24 mos.		x Post-op. x-ray treatment

tumour.

Group 3—carcinoma of a granulomatous pattern, a more mature variant of Group 2, showing lymphoid infiltration and mature Hassall's corpuscles.

Group 4—lymphoepithelioma.

Group 5—thymoma, a well encapsulated essentially benign tumour.

Group 6—carcinoma of adamantinomatous pattern, showing all transitions from a slowly growing tumour to a highly invasive anaplastic tumour.

Lowenhaupt proposes that lymphocytic infiltration is the only common histological finding in those tumours associated with the clinical syndrome of myasthenia gravis. From the opposite aspect, Keynes¹¹ has reported that two investigators, Dr. Collins of Sheffield and Dr. Bratton of the L.C.C. service, believe they are finding in most of the glands removed from myasthenic patients (not thymic tumours) an abnormal development of foci of clear cells resembling the so-called germinal centers of lymph nodes.

Myasthenia Gravis—It has been known for many years that there is some relationship between the thymus and myasthenia gravis. Approximately 10% of patients with myasthenia have thymic tumours. Conversely, of patients with thymic tumours, 30 to 40% have myasthenia gravis. Evidence now exists that removal of the apparently normal thymus from myasthenic patients will result in complete, or nearly complete remission of symptoms in 65% of cases. Keynes,¹³ Ross.¹⁸

As early as 1912, Saurebruch attempted to remove the thymus from a myasthenic patient. The first recorded operation for benign thymic tumour in a patient with myasthenia, was done by Blalock,^{2,3,4,5} in 1936. In the Winnipeg General Hospital series of operations for thymic tumours, there were 4 out of 10 patients who first presented with myasthenia gravis. In addition to this tumour series at least three other patients without tumours have had thymectomies for myasthenia gravis done here. The author has had the opportunity to do one of these, for Dr. R. T. Ross, neurologist. These cases are not the subject of this paper, but interestingly enough, our lady had a thymus weighing 41 grams and containing an unusual number of germinal centres.

Operation—Saurebruch, in 1912, and Haberer in 1917, had attempted thymectomies through the neck, but of necessity, these must have been incomplete. Blalock, as mentioned before, did the first recorded operation for benign thymic tumour in a myasthenic patient. This was in 1936. As far as one can determine, this was the first successful excision for any type of thymic tumour. The details of operative technique are beautifully described and illustrated in the classic paper by Keynes¹¹ and need not be repeated here. It is of interest to note that nine of the ten in our series were done by splitting the sternum and one done through a right thoracotomy.

Case Reports:

(1) C.B.—Age 36, female. This lady's first symptom was pain over her left breast in February, 1947. She came to hospital in May, 1947.

Table I

Gross Description	Histology	Lowenhaupt Group	Follow-Up
Large ovoid encapsulated cystic mass	Thymic cyst		Alive and well. No evidence of tumour, 7½ years post-operative.
Thymus with left lobe lobulated tumour. The tumour weighed 59 gm.	Thymoma	5	Alive and well. Off Prostigmine 7½ years post-operative.
Firm nodular, invasive tumour, non-resectable.	Malignant thymoma	4	Died of tumour, 11 months post-operative.
Firm encapsulated tumour.	Undifferentiated carcinoma	5	Alive and well. No evidence of tumour 5½ years post-operative.
Firm fixed non-resectable tumour with node involvement 1552 gm. segment of tumour excised.	Epidermoid cancer with fibroblastic reaction	6	Died of tumour 2 years and 2 months post-operative.
Circumscribed non-adherent tumour replacing one inferior pole of thymus. Tumour 40 gm., thymus 9 gms.	Malignant peritheliomatous type of thymic tumour	5	Died of myasthenia gravis, and no evidence of tumour, 1 year and 9 months post-operative.
Specimen 68 gms.; thymus 26 gms.; fat 40 gms.	Malignant thymoma	3	Alive with myasthenia gravis, and no evidence of tumour 3½ years post-operative.
Tumour invading pleura and pericardium; non-resectable. Segment of 42 gms. excised.	Malignant thymoma	2	Alive with myasthenia gravis, and no evidence of tumour 2 years, 4 months post-operative.
Encapsulated firm tumour, weight 152 gms.	Thymoma	5	Alive and well. No evidence of tumour 6 months post-operative.
Hard, flat disc-like tumour adherent to sternum and invading veins.	Epidermoid cancer with fibroblastic reaction	6	Alive with signs of tumour, 4 months post-operative.

Chest x-rays showed a mediastinal mass. There was no adenopathy or enlargement of liver or spleen. There was bloody fluid in the left chest and smears of this were negative for tumour cells. She received pre-operative radiation therapy with little, or no, change in the lesion. On July 24, 1947, the tumour was removed. It was a large ovoid encapsulated cystic mass; weight not recorded. The pathologist reported it as a cystic lesion of thymic origin. This patient has remained well.

(2) M.L.—Age 31, female. This patient developed myasthenia gravis in July, 1947 just prior to parturition. She received Prostigmine, 45 mgms., q 3 h, with partial relief. She was admitted in September, 1947, and chest x-rays showed a mediastinal mass. There was no adenopathy or enlargement of liver or spleen. On September 25, 1947, she was operated upon and the thymus containing a left lobe tumour was removed. The tumour itself weighed 59 grams. It was lobulated and had cystic spaces. The pathologist reported it to be a thymoma. The patient has done well. Her Prostigmine was first reduced and finally discontinued in October, 1953.

(3) M.R.—Age 27, female. This lady's tumour was discovered by routine chest x-ray in March, 1948. On subsequent questioning she admitted to slight symptoms of fullness in her neck and discomfort in the neck and left shoulder. Examination revealed a mild superior vena caval syndrome. There was no generalized adenopathy or enlargement of liver or spleen. On April 15, 1948, she was operated upon and a firm, nodular, invasive tumour was found replacing the thymus and attached to pleurae and pericardium. The tumour was biopsied but could not be excised. The pathologist reported malignant tumour of the thymus. The patient received subsequent radiation therapy with temporary regression of the mass. She died in February, 1949 of her tumour. No post mortem was obtained.

(4) H.W.—Age 62, female. This lady's tumour was discovered by a routine chest x-ray in May, 1948. She was asymptomatic and was observed for a year. In April, 1949, the mass increased slightly and she was admitted to hospital. There was no adenopathy or liver or spleen enlargement. On April 16, 1949, the anterior superior mediastinum was explored and an encapsulated tumour removed from the thymic site. The weight was not recorded. The pathologist reported it to be an undifferentiated carcinoma, suggestive of thymic origin. She received post-operative radiation therapy, and is now alive and well.

(5) R.S. Age 33, male. This man's tumour was discovered by a routine chest x-ray in January, 1949. He was asymptomatic. There was increased dullness in his left chest anteriorly. There was

no adenopathy or enlargement of liver or spleen. On January 29, 1949, the anterior superior mediastinum was explored. A large firm fixed tumour was found with bilateral extensions into the pleurae. An enlarged internal mammary node was seen and biopsied. A segment of tumour, weighing 1552 grams was excised. The pathologist reported the tumour to be a primary malignant tumour of the thymus growing mainly as a fairly well differentiated epidermoid carcinoma, with a marked fibroblastic reaction. The node biopsy was reported metastatic tumour. He received subsequent radiation therapy. He died of his cancer in March, 1951.

(6) E.V.—Age 52, male. This man developed myasthenia gravis in May, 1950. Chest x-ray in July, 1950 was reported negative. He received an unrecorded amount of Prostigmine with benefit. Repeat x-rays in November, 1950 showed an anterior superior mediastinal mass. On November 25, 1950, he was operated upon and a firm, circumscribed, non-adherent thymic tumour was found replacing one inferior thymic pole. The thymus with the tumour was excised intact. The whole weighed 49 grams, the tumour alone weighing 40 grams. The pathologist reported malignant peritheliomatous type of thymic tumour. The patient received post-operative radiation therapy. He continued to require Prostigmine, dosage not recorded, and died of myasthenia gravis, without evidence of recurrent tumour in August, 1952.

(7) E.T.—Age 34, male. This man developed myasthenia gravis in December, 1950. He was seen here in April, 1951 when chest x-ray was negative, but fluoroscopy with deep inspiration showed a spherical mass in the anterior mediastinum. On May 10, 1951, a thymectomy was done. The specimen weighed 68 grams and readily separated into fat, weighing 40 grams and thymus weighing 28 grams. The pathologist reported malignant thymoma. The patient received post-operative radiation therapy. His myasthenia gravis persisted and he continued his Prostigmine. He is now alive with myasthenia gravis, but without evidence of tumour.

(8) M.T.—Age 36, male. This man developed myasthenia gravis in June, 1949. Serial chest x-rays showed no mediastinal mass and he was treated with 150 mgms. of Prostigmine daily with partial relief. In August, 1952, a film revealed a superior mediastinal mass. On September 15, 1952, he was operated upon and a tumour found adherent to the pericardium and aorta. The tumour was incompletely excised. The specimen weighed 42 grams. The pathologist reported — malignant thymoma. The patient received radiation therapy. He continued to need Prostigmine. He is alive with myasthenia gravis and without evidence of tumour.

(9) A.R.—Age 57, male. This man's tumour was discovered by a routine chest x-ray in May, 1954. The records hint that review of a 1951 film showed the lesion which was missed at that time. He was asymptomatic and had no signs. On July 2, 1954, a right thoracotomy was done and an encapsulated mediastinal tumour weighing 152 grams was removed intact. The pathologist reported—encapsulated thymoma. Group 5—Lowenhaupt. The patient received no radiation therapy. He is now alive and without evidence of tumour.

(10) A.C.—Age 56, male. In September, 1952, this man developed pains in his neck and shoulders. In 1954, his head felt full, his face became flushed, and his neck veins became distended. Admission examination in September, 1954, revealed a superior vena caval syndrome. Chest x-rays failed to show a mediastinal mass. Bronchoscopy was negative. On September 30, 1954, the anterior superior mediastinum was explored. A very hard, infiltrating, disc-like tumour was found adherent to the overlying manubrium sterni and invading and constricting the great veins. The right pleura was opened and no tumour found in the lung. The lesion was biopsied, being non-resectable. The pathologist reported—infiltrating epidermoid cancer, growing in dense connective tissue. The patient received post-operative radiation therapy. He is well and working, with evidence of tumour in that his superior vena caval syndrome is only partially relieved.

Discussion—Ten patients have been operated upon for thymic tumours in the Winnipeg General Hospital since July, 1947. There were six males and four females. Their ages ranged from 27 to 62 years, the median age being 36 years. Four of the ten patients had no complaints and their tumours were discovered by routine chest x-rays. Four presented with myasthenia gravis. One presented with chest pain, and one presented with a superior venal caval syndrome. One of the asymptomatic patients picked up by routine film did prove to have a mild superior vena caval syndrome, on examination.

The time from onset of symptoms, or discovery of the tumour by routine films, until surgery, ranged from one to thirty-nine months. For those six patients not myasthenic, the time interval until surgery ranged from one to twenty-four months. The four myasthenic patients had time intervals of two to thirty-nine months. The two patients that are dead of their tumours had time intervals from their pick up by routine film until surgery of one month or less. In these two, the tumours were non-resectable. The third death was a patient with myasthenia gravis for six months prior to surgery. At operation, the encapsulated tumour appeared to have been completely removed. This man died of myasthenia without evidence of

residual, or recurrent, tumour, 21 months after surgery.

At operation, six of the patients had apparently complete excision of their tumours. Three of these six "favorable" cases received post-operative radiation. Five of the six are alive without evidence of tumour. One of the six died of myasthenia gravis 21 months after surgery, but without evidence of tumour. The five survivors in this "favorable" group range from 7½ years to 6 months since surgery.

The four patients whose tumours were non-resectable all received post-operative radiation. Two died of their tumours, 11 and 26 months after operation respectively. Two of these "unfavorable" patients are alive, one 28 months, the other 4 months since surgery.

The six "favorable" cases with tumours apparently completely excised and no evidence of recurrence, had varied histological types of tumour. There was one thymic cyst; one thymoma, one undifferentiated carcinoma; one malignant thymoma; one encapsulated thymoma Group 5, Lowenhaupt; and one malignant peritheliomatous type of thymic tumour. As mentioned above, five of the six are alive and well, one died of myasthenia with no evidence of recurrent tumour.

The four non-resectable tumours were biopsied and reported as follows: one malignant tumour of the thymus; one malignant thymoma; two epidermoid carcinomas with marked fibroblastic reaction.

Conclusion—Ten patients were operated on for thymic tumours, seven are living and three are dead. Of the seven living, six have no evidence of tumour, one is living with evidence of tumour, three of the six are over five years from operation. Three patients are dead, two died of their cancer, one died of myasthenia gravis one year and nine months after surgery without evidence of recurrent tumour.

No one of the six patients whose tumours were resectable has had any sign of residual or recurrent tumour. This indicates, of course, that the aggressiveness of any particular tumour is the single most significant factor in prognosis.

In considering the significance, if any, in the delay time, that is, from onset of symptoms until surgery, one is impressed by the wide range in growth rate of individual tumours. Cases 3 and 5 were picked up by routine film when they were asymptomatic. They were operated upon within a month; yet both proved non-resectable and both are dead of their tumours. Case 9 was picked up by routine film, proved resectable, and is living and well. Case 4 was picked up by routine film and was observed for a year. Fortunately, when she was finally operated upon, the tumour was still resectable and she is free of disease over five years.

The varying growth rate of individual tumours in any series is so great that attempting to compare the times of delay and the end results is valueless. The important conclusion from our data is that if one can operate on a particular patient while his tumour is still resectable, the prognosis is excellent. If one observes a mediastinal tumour, one is risking the patient's chance for a cure.

Acknowledgments — I wish to thank my colleagues at the Winnipeg General Hospital, and especially Dr. M. B. Perrin, Dr. C. B. Schoemperlen and Dr. J. W. Rennie for permission to include their cases. Dr. D. Penner has been kind enough to review the slides and classify the tumours by the grouping of Lowenhaupt. Dr. McGuire and her staff in Medical Records and Miss Macdonald and her staff in Tumour Registry have been most helpful and co-operative.

Abstracts from the Literature

Dysphagia of Transitory Type Produced by Hypertrophic Spurs on Cervical Vertebrae. H. Stephens, W. L. James. *Ann. Int. Med.*, 41: 823-828, 1954 (Oct.)

Dysphagia may be produced by protruding spurs of cervical hypertrophic arthritis of the spine. The lesion may be demonstrated by barium swallow with the cervical spine slightly extended. It is not necessarily a part of severe generalized hypertrophic arthritis. It is an apparently comparatively benign lesion, of the many lesions causing dysphagia. Two cases are reported, with x-ray illustrations.

A. G. Rogers.

Cerebral Vascular Diseases: Their Significance, Diagnosis and Present Treatment, Including the Selective Use of Anticoagulant Substances. I. S. Wright, E. McDevitt. *Ann. Int. Med.*, 41: 682-698, 1954, (Oct.)

It is important to determine whether neurologic signs of an intracranial lesion are due to hemorrhage, local thrombosis, embolism from a fibrillating heart, aneurysm of a cerebral artery, or a tumour. With normal blood pressure and a regular cardiac rhythm, 60-85% of deaths due to cerebral vascular diseases are probably related to thrombosis, with hemorrhage and embolism accounting for the rest. An irregular cardiac rhythm favors the diagnosis of cerebral embolism. With hypertension, hemorrhage becomes more common but thrombosis still continues to dominate.

Spinal puncture is of great value. Xanthochromic or bloody fluid indicates hemorrhage.

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Clear fluid, with other factors, may lay a fairly sound basis for the diagnosis of thrombosis or embolism.

Carbon dioxide inhalations increase the blood flow to the brain, but final proof is wanting, that the inhalations are valuable clinically. Final evaluation of stellate ganglion block is yet to be done. Anticoagulants may (a) prevent propagation of the original thrombus, (b) prevent thrombi in other vessels, (c) encourage disintegration of the thrombus. Risks include: (a) the aggravation of hemorrhage, when cerebral thrombosis is diagnosed by mistake, (b) the aggravation of hemorrhage in a cerebral infarct, (c) the risk of hemorrhage elsewhere in the body.

57 patients during a period of 795 patient months before beginning anticoagulant therapy experienced 205 thrombo-embolic episodes, 81 of which were cerebral in location. After anticoagulants were begun, during a period of 1,162 patient months, these patients experienced 23 thrombo-embolic episodes, 6 being cerebral. The reduction appears to occur in emboli from thrombi in hearts of patients with rheumatic heart disease or with acute myocardial infarcts. A reduction in the incidence of recurrent thromboses in cerebral arteries was noted. The risk of hemorrhage in the treatment of these diseases with anticoagulants is present, but not excessive. Further studies are underway to accumulate additional evidence regarding the value of anticoagulants in the treatment of cerebral vascular accidents.

A. G. Rogers.

Obstetrics

The Management of Pregnancy in the Cardiac Patient

Leon Rubin, M.D., M.R.C.O.G.

Heart disease during pregnancy has in recent years become one of the leading causes of maternal mortality. This is not because there are more deaths from heart disease in pregnancy, but is due to the fact that the maternal mortality for cardiac patients in pregnancy has not decreased *pari passu* with the mortality due to other causes. In the United Kingdom:

In 1935 there were 1,748 maternal deaths, of which 85 were due to cardiac disease, an incidence of 5.1%.

In 1949 there were 509 maternal deaths, of which 58 were due to cardiac disease, an incidence of 11.4%.

The number of maternal deaths decreased by 71%, but the fall in maternal deaths due to cardiac disease was only 35%. In other words, although the mortality rate from causes as toxæmia, hæmorrhage, and infection has dropped markedly, heart disease still claims a relatively large number of victims. Recent surveys show that cardiac conditions are now responsible for about 25% of all maternal deaths, and in some centres are the most important causes of maternal death. As a result the management of pregnancy in the cardiac patient assumes an ever increasing importance.

Physiology:

It is not intended here to discuss the physiology of the heart in any detail, but some important factors must be considered. In pregnancy there is an increased area of circulation in the body. This increase consists chiefly of the utero-placental circulation, but includes also the breast and other organs. It is estimated that one-sixth of the total blood volume is present in the utero-placental circulation, which acts as a large shunt. This fact is important clinically in two ways. First, it imposes a greater strain on the heart during pregnancy; second, this large amount of blood is thrown back into the general circulation after the delivery of the placenta and then imposes a sudden strain on the heart.

During pregnancy the heart meets this added strain not by an increase in pulse rate but by an increase in cardiac output. The latter increases from the 12th week of pregnancy to the 28th week; it then remains steady until the 34th week, and then gradually falls till term. The peak load on the heart, then, is from the 28th to the 34th week of pregnancy and decreases toward term. This

is important clinically as it has been shown that interruption of pregnancy after the 12th week places a greater strain on the heart than does normal labour at term. It is apparent also that premature induction of labour would serve no useful purpose since the strain on the heart decreases toward term.

The healthy heart adjusts to this increased burden because of a good reserve. If the cardiac reserve is impaired, signs of failure may appear early. If heart disease is present with no or little functional impairment, there will be no symptoms, and pregnancy and labour may be uneventful as in perfect health. Sometimes unknown heart disease may become apparent during pregnancy because of the added strain.

The commonest etiological factor by far, in women of reproductive age, is rheumatic heart disease, and the commonest lesion is mitral stenosis.

Diagnosis:

The diagnosis of cardiac disease during pregnancy may be very difficult because the signs and symptoms referable to the cardio-vascular system in pregnancy resemble those of cardiac disease.

Dyspnea is common in pregnancy, but is rare before 28 weeks.

Tachycardia is usual, but is significant if the pulse rate is over 100 in spite of rest.

Oedema is so common as to be of no diagnostic value. This applies also to distension of the cervical veins.

Basal crepitations occur occasionally in pregnancy but disappear after a deep breath. If they persist, further investigation is warranted.

Enlargement of the liver is of little value and is difficult to diagnose in late pregnancy.

Changes in the heart sounds, and the presence of murmurs occur often and are not of much significance. The exception is the presence of a diastolic murmur.

Cyanosis and hæmoptysis are never normal and must be investigated.

Fainting attacks occur occasionally in pregnancy. These people are usually hypotensive and almost never have heart disease.

X-ray—The heart lies transversely in pregnancy and appears to be hypertrophied. There is still disagreement as to whether the normal heart hypertrophies in pregnancy.

Cardiographic changes occur in pregnancy. In standard lead III there is a deep Q wave and inversion of the T wave.

It is apparent that the diagnosis of actual cardiac disease during pregnancy may be most dif-

ficult, and even after thorough investigation is often uncertain.

Diagnosis:

This is the first and most important factor to establish, and in this respect the functional grading of the heart is of great value. It is not the lesion present which matters so much, but the actual response of the heart to effort. If the state of the myocardium is satisfactory, the prognosis is usually good and vice versa. All cardiac patients during pregnancy should be graded according to the classification of the American Heart Association:

Group 1. Patients with organic heart disease but with no limitation of physical activity.

Group 2. Patients with organic heart disease with slight limitation of physical activity. These people can perform ordinary housework.

Group 3. Patients with organic heart disease with marked limitation of physical activity. Only light duties are tolerated.

Group 4. Patients with organic heart disease with dyspnea at rest.

Groups 1 and 2 are unlikely to have trouble during pregnancy; Groups 3 and 4 are very likely to have trouble during pregnancy. Jensen reviews a series of 1,428 cases of heart disease during pregnancy with the following results:

Groups 1 and 2—Maternal mortality $\frac{1}{2}$ of 1%

Group 3—Maternal mortality 5%

Group 4—Maternal mortality 22%

He concludes that patients in Group 5 should have their pregnancies terminated.

While the functional grading of the heart is the most important factor in the prognosis, other factors must be considered. A history of previous heart failure is significant. Any patient who has been in failure prior to pregnancy, or in a previous pregnancy, is more likely to go into failure in any additional pregnancy because of the added strain. In many centres a history of failure prior to pregnancy is an indication to terminate pregnancy. Fibrillation is an important sign, as it indicates myocardial damage. Enlargement of the heart is a bad prognostic sign—the less the enlargement, the better the outlook. Subacute bacterial endocarditis is a rare but definite risk. The age of the patient is important. As rheumatic fever is a progressive disease in which the condition of the heart deteriorates with age, it follows that the older the patient, the greater is the danger. It is estimated that over the age of thirty-five, the risk is doubled. The number and spacing of the pregnancies is also important. The patient who is able to space her pregnancies and obtain sufficient rest between them will have a better prognosis than the patient whose pregnancies follow one another quickly. The risk rises steeply with the number of pregnancies, particularly after the third preg-

nancy. Social factors modify the prognosis. The patient who is well-to-do, and can afford help with her housework and with the new baby, will do better than her sister who has to carry the extra load by herself. It is evident, then, that all these factors must be weighed at the onset of pregnancy, and the patient graded according to the classification of the American Heart Association, so that the best management may be undertaken.

Management:

Before discussing the management, a few generalized statements may be made:

1. Most of these cases do well.

2. If the patient is in failure, treat the failure and not the pregnancy.

3. Decide at the outset whether or not to terminate the pregnancy. In general, the cases which should be terminated are those in Group 4 of the classification of the American Heart Association (mortality 22% in pregnancy), and those who have a history of failure prior to pregnancy. If termination is decided upon, it should be done before the end of the third month and the vaginal route should be used. After the third month it is necessary to empty the uterus via the abdominal route and it is preferable to allow the pregnancy to continue rather than to use this method, as it has been shown that this operation places more of a strain on the heart than does the actual pregnancy and confinement. If failure occurs in the first three months, treat the failure and terminate the pregnancy. If failure occurs after the third month, treat the failure and carry on with the pregnancy. Never empty the uterus during failure. Treat the failure first, and then, if necessary, terminate the pregnancy.

If the decision has been made to carry on with the pregnancy, there are three keystones of ante-natal care.

(1) Rest. This is most important. If necessary periods of hospitalization should be insisted on, in the more severe grades. This is particularly important between the 28th and 34th week.

(2) Avoid infection, particularly respiratory infections, as these are liable to precipitate pulmonary oedema.

(3) Correct anaemia and avoid toxemia. Salt must be restricted.

Labour:

Induction of labour has no place in the management of these cases. Since the strain on the heart lessens after the 34th week, there is nothing to be gained from this procedure.

Caesarian section has practically no place in the management of these cases, and should be used only for some obstetrical complication such as disproportion, placenta previa, diabetes, etc. Caesarian section should never be used simply to sterilize the patient. If sterilization is considered necessary,

it should be done later, after the patient has been delivered via the vaginal route. With Caesarian section these cases have more shock, there is a greater risk of infection, and embolic phenomena are more frequent. The poor ventilation of the lung bases post-operatively, with the pulmonary congestion present, predisposes to the danger of atelectasis.

Haig and Gilchrist, reporting on a series of cardiac patients with severe disease (grades 3 and 4), show the following figures:

102 patients delivered by Caesarian—maternal mortality 12%.

277 patients delivered vaginally—maternal mortality 4%.

Cardiac patients have been traditionally considered to have easier labours. This has been ascribed to several factors. First, labour is often early; and second, dilatation and stretching of the soft parts and pelvic viscera occurs more readily because of the venous congestion and increased vascularity of these parts. Recent work, however, has questioned this belief, and the matter must be considered unsettled.

During the first stage of labour one must ensure sufficient rest. Adequate sedation must be used, and in this respect demerol, hyoscine, and if necessary, morphine, should be used. In the second stage the avoidance of bearing down efforts by the patient is important. As soon as the cervix is fully dilated and the head is at or near the perineum, outlet forceps should be employed and an episiotomy performed. Local anesthesia is of value in the perineum. General anesthesia may be employed, but should be used sparingly, and a high content of oxygen maintained no matter what anaesthetic agent is decided upon. After the placenta has been delivered and the uterus contracts down, the blood in the utero-placental circulation returns to the general circulation. This is estimated to be one-sixth of the total blood volume, and as it is rather suddenly thrown back, so to speak, it imposes an additional strain on the heart. For this reason, some authorities advocate allowing the patient to bleed a little more freely after the delivery of the placenta—a sort of vaginal venesection. In this respect, it is inadvisable to use oxytocics in these patients excepting those cases in which the bleeding is too profuse. An older method of reducing this added strain on the heart was to place a sand-bag on the abdomen after delivery of the placenta.

The most critical time is the immediate post-partum period. These cases have a tendency to develop pulmonary oedema and acute heart failure in the first twenty-four hours after delivery and must be observed carefully for several days. Morphine or a barbiturate should be given immediately after delivery and adequate rest ensured. If failure does occur during or after labour, dioxin, morphine, oxygen, aminophylline, and venesection may be used. The routine use of penicillin during labour is advocated for these cases; this is to prevent the development of subacute bacterial endocarditis.

Remote Prognosis:

The occurrence of pregnancy has no ill effect upon the course of the disease. If the patient survives the pregnancy and the puerperium, her cardiac condition returns to the status present prior to the pregnancy. Those cases which deteriorate do so because of age and the natural course of the disease, not because of intercurrent pregnancy. The latter does not accelerate the cause of the disease.

Heart Surgery:

In recent years heart surgery has come into prominence, and many cases previously believed to be hopeless have been rendered capable of carrying on with pregnancy. Valvulotomy may be performed in pregnancy, particularly in the early months. In suitable cases, this procedure may be preferable to termination of the pregnancy.

Summary:

1. Most of these cases do well.
2. The most important factor in the prognosis is the functional capacity of the heart.
3. Very few cases require termination of the pregnancy. If necessary, this should be performed before the end of the third month.
4. Adequate rest, and the prevention of infection are the most important measures in prenatal care.
5. If failure occurs in pregnancy, treat the failure, not the pregnancy.
6. Induction of labour and Caesarian section per se have practically no place in the management of these cases.
7. During labour, adequate rest and sedation, the avoidance of bearing-down efforts, and a minimum of inhalation anesthesia are most important.
8. The first twenty-four hours post-partum are the most dangerous, but these patients must also have a long period of rest in the puerperium.

"Problems of the Newborn Infant"

A series of case reports and commentaries from the files of the Winnipeg General, St. Boniface and Children's Hospitals, illustrating factors which affect the survival of the infant during his first week of life.

SERIES II

Abruptio Placentae

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The Stillbirth and Neonatal Mortality Research Project at the Maternity Pavilion, Winnipeg General Hospital, and St. Boniface Hospital was begun April 1st, 1954. During the first nine months it has functioned at the General Hospital the diagnosis of "Abruptio Placentae" (premature separation of the normally implanted placenta) was made 16 times, an incidence of .5%.

8 of these cases resulted in stillbirth and 1 in neonatal death. The neonatal death was a baby weighing 750 grams at birth, 28 weeks gestation, which was delivered following a long period of vaginal bleeding. 1 case was twin birth, the first twin being stillborn but the second survived. Therefore 8 babies survived. Of these 8 cases, cesarean section was performed three times on the indication of abruptio. The remaining 5 cases were delivered vaginally following careful watching on the ward (1 case was about to be sectioned when it was found that vaginal delivery was imminent. This case is reported below).

Abruptio placentae was formerly called "Accidental Hemorrhage Revealed and/or Concealed", but in recent years this terminology has been dropped from the literature. Some centres state that the association with toxemia is as high as 95%, but that is not the incidence now generally accepted, although toxemia associated with the condition is usually quite high. Many non-toxemic cases are perhaps marginal sinus rupture. Unsatisfactory implantation of the ovum in the endometrium is undoubtedly a major factor, as circumvallate placenta and battledore insertion of the cord are frequently reported in association with the condition.

Diagnosis

In contrast to placenta previa the placenta is not implanted in the lower uterine segment, hence the term "premature separation of the normally implanted placenta".

Bleeding with placenta previa is generally bright as the blood has a direct means of escape, and is usually painless to the mother. In contrast the bleeding of abruptio is darker, as the blood is trapped behind the placenta before escaping, and

is usually accompanied by pain, the severity of which depends on the extent of separation and the amount of retention of blood. Nevertheless differential diagnosis can be extremely difficult in some cases. Therefore the following rules should be carried out in all cases of antepartum hemorrhage in the third trimester.

1. Immediate hospitalization and treatment of shock, including grouping and matching for blood. Shock may be present despite little, if any, external bleeding.

2. No rectals or enemas at any time.

3. Vaginal examination only when

- (a) Diagnosis is made and it is decided to do surgical rupture of membranes or
- (b) Where diagnosis is uncertain but placenta previa is suspected (here it is best to do the vaginal examination in an operating room prepared for cesarean section).

4. Placentogram in mild cases, particularly if placenta previa is suspected.

It is with abruptio placentae that the condition of afibrinogenemia in the mother is most frequently associated. (The normal blood fibrinogen is activated by a thromboplastin presumably released by the injured placenta and deposited as fibrin in minute amounts throughout the blood stream. The normal blood fibrinogen level is approximately 300 mgms. percent. If the reaction is great enough to lower the level to 90 mgms. or less, bleeding from any slightly traumatized area will occur). This condition should be checked for frequently in all cases of abruptio. All one requires to do is withdraw some venous blood, preferably in a keidel tube, and check the length of time it takes for the blood to clot, and also to observe the appearance of the clot. Lower nephron nephrosis is also a complication of abruptio and seems to occur more readily than one would expect from the extent of shock and blood loss. This is probably due to the fibrin deposits in the kidney vessels. The condition should be watched for carefully if there is a sudden gross albuminuria associated with the abruptio.

Ernest Page of University of California School of Medicine in an article "Abruptio Placentae—Dangers of Delay in Delivery" gives a clinical classification and recommended principles of delivery which is now widely quoted and followed.

Page Classification

Grade 0 — Clinically unrecognized before delivery. Diagnosis based on examination of placenta.

Grade 1 — External bleeding only, or mild uterine tetany but no evidence of maternal shock.

Grade 2 — Uterine tetany, ordinarily with uterine tenderness, possibly external bleeding, fetal distress (or death) but no evidence of maternal shock.

Grade 3—Evidence of maternal shock or coagulation defect, uterine tetany, and intrauterine death of the fetus.

Treatment (Quoted from Page's article)

Grade 1 severity—Replacement of blood loss, rupture of membranes or induction of labour in some cases near term.

Grade 2—Same as Grade 3 except that coagulation defects are less common, shock is absent, and if fetal heart tones are present, an immediate cesarean section is justified in the interests of the baby as well as the mother.

Grade 3—

1. Immediate examination of blood for hemoglobin, hematocrit, clotting time, clot observation, rapid estimation of plasma fibrinogen, typing and crossmatching.

2. Prompt vaginal examination and rupture of membranes. (This assumes that the diagnosis is confirmed but the combination of maternal shock, uterine tetany and absent fetal heart tones is essentially pathognomonic of a major separation with concealed hemorrhage). Membranes are ruptured irrespective of the condition of the cervix, because the purpose is to lessen the intrauterine pressure and reduce the chances for continued intravenous auto-injections of potentially lethal tissue extractives. By the same token, oxytocic stimulation is probably contraindicated.

3. Prompt replacement therapy with fibrinogen if indicated, and ample quantities of fresh whole blood. This must be done before cesarean section is undertaken. Having restored blood and corrected existing coagulation defects, any further delays in delivery are dangerous.

4. Based on the amount of dilatation of the cervix, presence or absence of labour, degree of shock remaining, decision is made as to which mode of delivery will permit termination of the pregnancy within the next 2-4 hours. Unless the cervix is more than half dilated, cesarean section is ordinarily the method of choice.

Four selected case histories taken from the files during the period referred to, April 1st - December 31st, 1954, are presented. In two cases the infants survived, in two cases the infants were stillborn.

1. Mrs. "A"

Age 27, P.O. G.1. Normal prenatal course. B.P. on admission 110/72. No albumin on admission urine specimen. Admitted at term because of bleeding for one hour, which did not stop. Bleeding was fairly heavy and steady for the next two hours. Consultation was obtained and immediate

cesarean section was performed as the fetal heart was good. A normal female child, 7 lbs. 9½ ozs. (3550 grams) was delivered. There was a large blood clot behind the placenta and old blood present. Post-operative hemoglobin was 54% (8.4 grams). In this case the membranes were not ruptured before the cesarean section and there is no record of checking on blood coagulation. Post-operative course was uneventful for mother and baby.

2. Mrs. "B"

Age 33. P.1. G.4. 37 weeks pregnancy. A history of intermittent bleeding throughout pregnancy since 1st trimester, moderate in amount. She was admitted because of ruptured membranes, there was no bleeding at the time. Maternal B.P. 110/70. Admission urine negative for albumin. Fetal heart good. Because of the history of bleeding and fetal head remaining high a placentogram was done on the day of admission which was reported: "Placenta lies anteriorly and on right side. It does not appear to have a low implantation despite the fact that the head is situated a little to the right of mid line".

At 1.30 a.m. the next morning the patient began bleeding moderately. She was in early labour. She did not complain of pain but it was noted that the uterus did not relax well between contractions. Because the placentogram had been done and the head had descended into the pelvis a low implantation placenta previa was ruled out. Patient was immediately grouped and matched. Fetal heart and maternal condition were watched carefully. (Membranes were already ruptured before admission).

At 7.30 a.m. the patient had not stopped bleeding. As the fetal heart was good and the uterus relaxing well between pains, it was felt that section was not indicated at this time, but at 8.00 a.m. another brisk hemorrhage occurred, so preparation for cesarean section was made. However, just before the patient was taken to the O.R. she was found by rectal examination to be fully dilated. Consequently she was taken to the Case Room and delivered of a male, 4 lbs. 7½ oz. (2100 grams) which has done well to date and has shown no evidence of retardation.

The placenta showed considerable areas of fibrosis, particularly around the periphery. In addition there was an area of recent hemorrhage involving about 25% of the area of the placenta and a clot totalling about 250 ccs. attached to the membranes.

There was no check on the patient's blood clotting time done. There was no further bleeding on the case room table, but 24 hours later the patient ran a temperature of 100.6°, complained of lower abdominal pain and passed a large blood clot.

3. Mrs. "C"

Age 30. P.2, G.4. She had toxemia in her second pregnancy and required induction of labour because of it. Following this she had a chronic hypertension, B.P. usually 160/90. Albumin negative.

At 29 weeks of pregnancy she passed a little blood. Placentogram at this time showed placenta high up and probably on the left side. She continued to have frequent prenatal examinations. During this period there was no change in the B.P. and no albuminuria. At 34 weeks she was admitted to the Hospital at 11.50 a.m. because of steady pain in the back which came on suddenly at 8.00 a.m. There was no bleeding per vagina. On admission catheterized urine was 0.6% albumin, B.P. 139/90. She had a fair amount of abdominal and low back pain. The uterus felt moderately firm. Fetal heart sounds were heard 100 per minute and were muffled. She was given Demerol. Two hours later her pain was worse and despite the absence of vaginal bleeding a diagnosis of abruptio placentae was made on the basis of her pain and distress and a firm uterus. Fetal heart sounds were still audible. The patient was nauseated and vomiting. Hemoglobin was 58%. Consultation was obtained immediately and it was decided that the risks to the mother, in view of her past history, and her present condition, outweighed the chances of obtaining a live infant by immediate cesarean section. It was therefore decided to do a surgical induction immediately and start emergency blood transfusion.

At this point 5 ccs. of blood were withdrawn to check the blood clotting time. The blood did not clot. When the induction was performed old blood escaped from the uterus, but there was no fresh bleeding.

She was watched carefully, transfusions continued and in 4 hours she delivered a male stillborn, 5 lbs. 2 ozs. (2400 grams). The placenta was delivered followed by two large blood clots. About one-half of the placenta had separated, the clots had made a shallow depression in the separated area of the placenta. There was a small perineal laceration which began oozing blood and could not be stopped by pressure or suturing. As blood samples would still not clot one gram of commercial fibrinogen was given in 50 ccs. of water intravenously. The blood lost during delivery remained fluid. Bleeding was then controlled and in one hour the clotting time was 7 minutes. Patient made an uneventful recovery. Postmortem examination of the fetus showed anoxic changes only.

In reviewing this history it will be noted that this patient went through the various stages of Page's classification from 1 to 3 in a very short

period of time. This indicates that if the fetus is going to be saved without jeopardizing the mother there should be no delay in hospitalizing the patient immediately and making early decisions as to delivery. Diagnosis in this case was hampered by the complete lack of vaginal bleeding until the membranes were ruptured.

4. Mrs. "D"

Age 22. P.O, G.1. This patient attended the Maternity Out Patient Department of the Winnipeg General Hospital. Until the day before admission at her 34th week in pregnancy, B.P. was stationary at 115/70 and urine was normal.

At 7.00 a.m. at home, she began to have steady abdominal pain, at 11.15 a.m. she began bleeding quite heavily per vagina while still at home. She was immediately admitted to Hospital. Her pads, clothing and a towel were soaked with blood. B.P. was 130/90. There was no albuminuria. The abdomen was quite firm. The attending obstetrician was unable to diagnose fetal position or hear fetal heart sounds. Maternal pulse was 104.

Consultation was obtained immediately. In view of the patient's condition and uncertainty of fetal heart sounds a conservative approach was decided upon, namely examination under anaesthesia and surgical induction. Blood transfusion was begun. Following preparation of the O.R. at 2.10 p.m. she was examined. There was no evidence of placenta previa on the sterile vaginal examination so induction was performed. 3 to 5 ozs. of fairly clear amniotic fluid was obtained.

The patient was watched carefully and at 9.00 p.m. delivered of a stillborn male, 2300 grams. There were many large, old blood clots adherent to the placenta which had been depressed in these areas. The placenta was circumvallate. The patient made an uneventful recovery. There was no bleeding from the tissues. No check on the blood clotting time had been made.

Postmortem examination of the fetus showed anoxic changes only. On pathological examination the placenta appeared normal.

It is the hope of the Committee that this brief review of the condition of Abruptio Placenta may help those who are suddenly faced with the problem to recognize it properly and effect immediate measures which may save the baby. It is felt that if all pregnant women are warned of the dangers of bleeding, especially if toxemia is present, and told to proceed immediately to hospital if bleeding should occur in the third trimester, then even less delay will occur in the management of these cases with perhaps a more happy outcome for the baby.

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Children's Hospital, Winnipeg

Ward Rounds

Edited by Wallace Grant, M.D.

Internes' Seminar — October 8, 1954

Heart Failure in Childhood — Part I

Review of the Children's Hospital Experience
1949-1953.

Dr. Ron Lauer

Heart failure in young children is frequently related to the presence of a congenital heart lesion. As more and more of these malformations become amenable to surgery, recognition and treatment of heart failure in children are increasingly important.

Causes of Heart Failure in Childhood

As in the adult, heart failure in childhood may be due to insufficient output of the right or left ventricle, or a combination of both of these factors. The factors causing or contributing to heart failure in childhood are set forth in Table I.

TABLE I

Factors Causing or Contributing to Heart Failure

A. Mechanical Overloading of the Heart

1. Increased Resistance to Ejection of Blood from the Left Ventricle

- A. Coarctation
- B. Subaortic stenosis
- C. Aortic Atresia
- D. Polycythemia
- E. Systemic Arterial Hypertension
 - i) Chronic pyelonephritis
 - ii) Chronic glomerulonephritis
 - iii) Congenital polycystic disease of the kidney
 - iv) Pheochromocytoma
 - v) Wilms' Tumor
 - vi) Cushing's Syndrome
 - vii) Lead poisoning
 - viii) Pink Disease

2. Increased Resistance to Ejection of Blood from the Right Ventricle

- A. Pulmonary stenosis (Tetralogy of Fallot)
- B. Pulmonary hypertension
 - i) Patent Ductus Arteriosus
 - ii) Atrial septal defect
 - iii) Abnormal entry of pulmonary veins
 - iv) Mitral stenosis

3. Excessive Demand for Output Affecting Left Ventricle Only

- A. Aortic regurgitation
- B. Mitral regurgitation
- C. Patent ductus arteriosus

4. Excessive Demands for Output Affecting Right Ventricle Only

- A. Patent septal defects, particularly of atria
- B. Eisenmenger's complex

5. Excessive Demands for Output Affecting Both Ventricles

- A. Arteriovenous fistula
- B. Anemia
- C. Excessive Blood Volume
 - i) Excessive administration of fluids
 - ii) DOCA & intoxication
 - iii) Acute nephritis
 - iv) Fever
 - v) Ventricular septal defects

B. Primary Myocardial Insufficiency

- A. Myocarditis
 - i) Rheumatic
 - ii) Diphtheritic
 - iii) Septicemia
 - iv) Acute Interstitial Myocarditis (Fiedler's)
- B. Endocardial fibroelastosis
- C. Aberrant Coronary vessel
- D. Paroxysmal Tachycardia
- E. Glycogen storage disease
- F. Idiopathic Hypertrophy
- G. Collagen disease
 - i) Acute disseminated lupus erythematosus
 - ii) Periarteritis nodosa



C. Impaired Cardiac Filling

- A. Excessive tachycardia or cardiac irregularities
- B. Adhesive pericarditis
- C. Primary decline in blood volume
 - i) External hemorrhage
 - ii) Loss of fluid or whole blood into body tissues
 - iii) Loss of water and electrolytes from GI tract, kidneys &
 - E.G.: Severe diarrhea
 - diabetic coma
 - iv) Primary loss of vascular tone: Neurogenic type
 - E.G.: vasovagal syncope
 - injury to nervous system: disease of spinal cord, section of sympathetic nerves, &c.
 - v) Primary Arteriolar and/or Capillary dilatation: Vasogenic type
 - E.G.: drugs — histamine, nitrites, &c.
 - liberation of histamine-like substances in injured tissue.

The causes of heart failure as seen at the Winnipeg Children's Hospital over the years 1949-53 are set forth in Tables II, III, IV and V.

TABLE II

29 Fatal Cases Under Two Years of Age

1. Patent Ductus Arteriosus — 3 cases.
2. Subendocardial Fibroelastosis — 5 cases.
 - (1) One Case — clinical diagnosis.
 - (2) One case — associated with aortic stenosis.
3. Congenital Heart Disease — not further diagnosed.
 - (1) Non cyanotic — 5 cases.
 - (2) Cyanotic — 2 cases.
4. Mongolism associated with Congenital Heart Disease — 4 cases.
5. Abnormal Entry of Pulmonary Veins.
 - (1) Into right auricle — 1 case.
 - (2) Into pulmonary veins — 1 case.
6. Fiedler's Myocarditis — 1 case.
7. Coarctation — 4 cases.
 - (1) Associated with Mongolism — 1 case.
 - (2) Associated with Hypoplastic Kidneys — 1 case.
 - (3) Associated with Fusion of Aortic Cusps — 1 case.
8. Pulmonary Atresia — 2 cases.
 - (1) Associated with P.D.A., I.A.S.D. and Tricuspid Stenosis — 1 case.
9. Eisenmenger's Complex — 1 case.

TABLE III

11 Non Fatal Cases Under Two Years

1. Congenital Heart Disease — not further diagnosed — 8 cases.
 - (1) 1 case associated with S.B.E.
 - (2) 1 case associated with Paroxysmal Tachycardia.
 - (3) 1 case occurred in a Mongol.
2. Inter-Ventricular Septal Defect — 1 case.
3. Inter-Auricular Septal Defect — 1 case.
4. Tetralogy — 1 case.

TABLE IV

5 Fatal Cases Over Two Years

1. Rheumatic Heart Disease — 2 cases.
2. Congenital Heart Disease — not further diagnosed — 2 cases.
 - (1) Non-cyanotic — 1 case. Occurred in a Mongol.
 - (2) Cyanotic — 1 case.
3. Chronic Nephritis — 1 case.

TABLE V

11 Non Fatal Cases Over Two Years

1. Rheumatic Heart Disease — 8 cases.
 - (1) 1 case associated with C.H.D.
 - (2) 1 case associated with S.B.E.
2. Congenital Heart Disease — 4 cases.
 - (1) Tetralogy — 1 case.
 - (2) Non diagnosed — 2 cases; 1 associated with R.H.D.
 - (3) I.A.S.D. — 1 case.

Signs and Symptoms of Heart Failure in Childhood

The signs and symptoms of heart failure of the above cases are shown in Tables VI, VII, VIII and IX.

TABLE VI

Signs and Symptoms of Heart Failure in Fatal Cases
Under Two Years of Age — 29 Cases

Fever	19
Increased Respiratory Rate	28
Pulmonary Adventitious Sounds	17
Cyanosis with Acute Illness	5
Clinically Enlarged Heart	9
Heart Enlarged in X-Ray	18
Tachycardia over 120/m.	26
Hepatomegaly	18
Significant Weight Gain	4
Frequent Pulmonary Infections	4
EKG Right or Left Ventricular Hypertrophy	4

TABLE VII

Signs and Symptoms of Heart Failure in Non-Fatal Cases
Under Two Years of Age — 11 Cases

Fever	6
Increased Respiratory Rate	8
Pulmonary Adventitious Sounds	4
Cyanosis with Acute Illness	1
Clinically Enlarged Heart	4
Heart Enlarged in X-Ray	9
Tachycardia over 120/m.	7
Hepatomegaly	5
Significant Weight Gain	3
Frequent Pulmonary Infections	2
EKG Right or Left Ventricular Hypertrophy	1

TABLE VIII

Signs and Symptoms of Heart Failure in Fatal Cases
Over Two Years of Age — 5 Cases

Fever	3
Increased Respiratory Rate	3
Pulmonary Adventitious Sounds	2
Clinically Enlarged Heart	3
Tachycardia — 120/m.	3
Hepatomegaly	1
Peripheral Oedema	1
Distended Neck Veins	1

TABLE IX

Signs and Symptoms of Heart Failure in Non-Fatal Cases
Over Two Years of Age — 11 Cases

Fever	7
Increased Respiratory Rate	7
Pulmonary Adventitious Sounds	4
Clinically Enlarged Heart	2
Heart Enlarged in X-Ray	10
Tachycardia — 120/m.	8
Hepatomegaly	5
Peripheral Oedema	4
Distended Neck Veins	2

It will be seen from these Tables that in children less than 2 years of age the signs and symptoms of heart failure vary somewhat from the usual adult pattern. In this age group heart failure resembles respiratory infection in that fever, tachypnoea, tachycardia and pulmonary adventitious sounds dominate the picture. The points which differentiate heart failure from respiratory infections are enlargement of the heart and hepatomegaly. (It will be seen from the Tables that the enlargement of the heart is most easily and accurately found by chest X-ray.)

In children over the age of 2 years the signs and symptoms of heart failure come more to resemble those seen in the adult, and distended neck veins and peripheral edema are not infrequent. The signs of respiratory illness may still predominate, but the finding of an enlarged heart and hepatomegaly again serve to differentiate the condition.

Congenital Cardiac Deaths at the Winnipeg
Children's Hospital (1949 - 1953.)

TABLE X

Congenital Cardiac Deaths — 26 Cases

Congenital Heart Disease — not specified	9
Subendocardial Fibroelastosis	5
Coarctation	4
Patent Ductus Arteriosus	3
Abnormal Entry of Pulmonary Veins	2
Pulmonary Atresia	2
Eisenmenger's Complex	1

Conclusions

From the above tables the following might be concluded regarding the cases of heart failure treated at the Children's Hospital over the past five years:

Preventable Deaths:

Patent Ductus Arteriosus	3
Coarctation	1
Pulmonary Atresia	1

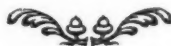
Possibly Preventable Deaths:

Congenital Heart Disease — Not further specified	7
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Preventable Deaths in the Future (?)

Abnormal Entry of Pulmonary Veins	2
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(To be concluded)



Radiotherapy

Progress in Radiotherapy

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Very soon after Roentgen's discovery of x-rays, nearly 60 years ago, the therapeutic possibilities of this new agent were being explored. Within a few months, cancer of the skin was treated with x-rays, and shortly they were applied to other forms of malignant disease and not a few benign conditions. At the same time the harmful effects of x-rays were not slow in making themselves known. Roentgen announced the discovery of x-rays on December 28th, 1895. By January, 1896, the first case of x-ray dermatitis of the hands was described; by April of the same year, epilation without dermatitis was noted, and within seven years, a report of the first case of cancer in an old x-ray produced ulcer was published.

I was made aware rather strikingly of the deleterious effects of x-rays of those early days, some eight years ago. Early in 1896, a Bond Street shop in London had imported a machine to amuse their customers with a demonstration of Dr. Roentgen's amazing rays. Two young men, who were brothers, were engaged for this purpose, one to turn on the machine and hold the fluorescent screen, and the other to place his right hand in front of the screen, so that the bones of his hand were displayed for all and sundry to see. In 1947, I saw these two brothers in the Radiotherapy Department of the Middlesex Hospital in London. The one had multiple basal and squamous cell carcinomata about his face, and the other had a severe radiation dermatitis with multiple squamous cell carcinomata of his right hand and forearm.

These harmful effects, which may not manifest themselves for a long time after exposure to radiation, have a direct bearing on my subject; for he who would use the new and more powerful radiotherapeutic machines and substances, must bear in mind these delayed effects of radiation, and must assess the risks involved on the basis of his past clinical experience, and the experimental data available.

Sixty years ago surgery was the only treatment for cancer, but it had little to offer to the more advanced cases. The search for a new agent that might salvage some of these imparted a great impetus to the development of radiotherapy as applied to cancer. To start with, both x-rays and radium were used quite empirically, but almost at once the nature of the radiation and its mode of action came under active investigation, and the concepts of quantity and quality of radiation became established. The importance of the physical factors involved was recognized by the pioneers

of radiology as early as the first few years of this century, e.g. a unit of dosage (although the "roentgen" was not established until 1928). The importance of filtration was recognized and even rotation therapy, which has come to our attention so much recently, was envisaged by Kohl in 1906.

Thus, while to start with, radiation therapy was administered haphazardly, very soon a scientific basis was established, at least for the physical aspects of this agent.

At first radiology dealt with tumours which were easily accessible, either by virtue of being on or near the surface of the body for treatment by x-rays, or the implantation of radium needles, e.g. carcinoma of the lip, or mouth, or in or near body cavities into which radium could be placed, e.g. carcinoma of the cervix uteri. However, the equipment in those early days was hardly suitable for the treatment of deep seated tumours, and only at the end of the first World War did equipment, operating at about 200 kv. become available. This type of equipment is still standard in most radiotherapy departments, and the results of treatment were so much better with these machines than with the equipment available earlier, that the optimistic belief arose that it would be possible to treat successfully most patients with malignant disease if only the penetrating power of x-rays could be increased manifold. Then, experimental machines were constructed operating at 400,000 up to one million volt, and thus the range of super-voltage therapy was reached.

The first million volt machine to be used clinically was constructed by Dr. Robert S. Stone at the University of California Hospital in co-operation with Professor Lawrence and Dr. Sloane in 1934, and it remained in use until 1948. By 1940, several machines operating at one to two million volts were available commercially. Since then new machines have been invented, such as the betatron, the cyclo-synchrotron, and the linear accelerator capable of producing x-rays or electron beams at energies of many million volts. The highest voltage, to my knowledge, is being generated at Stanford University in California, where a linear accelerator operates at seven hundred million volts. Other machines were invented which accelerate heavier particles such as protons or alpha particles. They are the cyclotron and the synchro-cyclotron.

Since the establishment of nuclear reactors, artificial radioactive substances have become available and have been incorporated into beam units. Cobalt 60 has been used most extensively for this purpose and when employed in the large

telecurie therapy units produces radiation roughly equivalent to x-rays generated at 3 million volts.

What, then, of the results obtained with this supervoltage equipment? The one to four million volt range has been in clinical use long enough to allow preliminary assessment, while the multi-million volt machines are still in the experimental stage.

Certainly, supervoltage therapy has not yet turned out to be the panacea for all malignant disease; but, it has undoubtedly many advantages, which have become well established.

Radiation therapy endeavours to deliver a quantity of energy to a volume of tissue large enough to encompass the tumour to be treated. As little normal tissue as possible should be irradiated, because of the destructive effects of x-rays on all tissues, in order to minimize the local reaction. In a superficial lesion this presents no problem, but in a deep-seated tumour a good deal of normal tissue is interposed between body surface and tumour, and this must be traversed by the x-rays beam. Supervoltage radiation provides greater penetration and a more sharply delineated treatment beam and, thus, makes possible radiation of a much smaller volume than the conventional high voltage therapy. Conversely, a sufficiently high tumour dose can now be delivered at a depth beyond the reach of 200 kv. x-rays. Apart from the local effect, x-rays absorbed in the body can cause a general reaction, particularly when large areas of the chest or abdomen are treated. This general effect, known as radiation sickness, is characterized by lassitude, nausea, vomiting and depression. Its occurrence depends on individual sensitivity, but also largely on the total volume irradiated and the total amount of energy absorbed in the body, and it will, therefore, be diminished by the use of supervoltage radiation.

I have already mentioned the more sharply delineated beam. This is due to the fact that the secondary radiations are scattered to a lesser degree sideways or backwards but rather more forwards. As the result, the effect on the skin is much reduced with supervoltage therapy. For instance, while at 200 kv. the maximum dose is received at the skin, at two or three million volts, this maximum is displaced several millimeters beneath the skin, while at 24 million volts the maximum effect does not appear for $3\frac{1}{2}$ centimeters deep to the skin. Thus, with supervoltage therapy severe skin reactions are no longer encountered. By the same means, when the scalp has to be irradiated, the degree of epilation is diminished and regrowth of hair occurs more quickly.

One further physical advantage lies in the fact that the differential absorption of x-rays in bone is greatly diminished in the supervoltage range

compared to two hundred to four hundred kv. x-rays, and, thus, the risk of bone necrosis or excessive radiation of bone marrow is reduced.

Deep-seated tumours then, preferably small in volume, are suitable for supervoltage therapy, and it is in these that most improvement in results can be expected. The pathology of these tumours may vary from moderate radio sensitivity to marked radio resistance. Of the former, transitional cell carcinoma of the bladder may be taken as an example. These tumours are known to be radio curable, and, if of small size and not too deeply infiltrating into bladder muscle, have been treated successfully by radon seed or radio active gold grain implants. However, in patients with tumours unsuitable for this form of treatment or for partial cystectomy, and who were subjected to conventional x-ray therapy exceedingly poor results were obtained. This was due to the fact that a sufficiently high dose could not be given to the affected volume without exceeding either the local or the general tolerance of the patient. At the Royal Cancer Hospital, London, a large number of patients suffering from bladder cancer have been treated for the past four years with supervoltage x-rays, and the immediate results have been markedly improved compared to the treatment in the past. Hardly enough time has elapsed to speak in terms of cure or percentage cure rate, but an appreciable proportion of patients, including many beyond any form of surgical treatment, are alive and symptom free for periods from one to three years.

So far the treatment of tumours known to be radio resistant such as the adenocarcinoma of the stomach or large bowel has been unsatisfactory. This may be because patients are frequently referred for radiotherapy only when massive recurrence is present. Perhaps pre-operative irradiation of the doubtfully operable case particularly in carcinoma of the rectum will reduce the number of recurrences.

Other tumours particularly suitable for supervoltage therapy are those situated close to, or shielded by bone, such as tumours in the accessory nasal sinuses, the buccal cavity and intracranial tumours. As mentioned before, the danger of bone necrosis is lessened and the bone does not interfere with an adequate tumour dose being given. Moreover, with the sharply defined beam, it is easier to avoid or minimize radiation to sensitive structures such as the eye.

My own experience with supervoltage radiation, particularly in brain tumours, has been very encouraging, and again very good early results have been obtained, even in tumours hitherto considered unsuitable for radio-therapy such as craniopharyngiomas.

Carcinoma of the bronchus has been treated with million volt x-ray therapy since 1937, but the

general experience as far as cure is concerned has been disappointing, although palliation is achieved more easily.

Supervoltage, then, puts at our disposal a means of delivering high doses of radiation to deeply seated tumours without upsetting the patient's general condition or causing severe local reactions in the normal tissues about their tumours. It means that the patient can support his treatment a great deal more easily, which matters greatly to the patient in whom cure is attempted and who has to undergo a prolonged and extensive course of treatment, as well as to the patient beyond hope of cure who can now receive a large dose of palliative radiation in a few days without adding to his discomfort or necessitating a long stay in hospital. Of course, not all patients are suitable for or require supervoltage therapy, and many can be treated equally well or better at lower voltages.

I should now like to turn to another development in radio-therapy, namely the application of radioactive isotopes. The use of radium, a naturally occurring radio active isotope is well known in the form of needles or tubes for interstitial or intracavitary radiation. Used empirically in the early days, a set of rules was established by Patterson and Parker in 1934 for radium to be applied in definite geometrical arrangements, which made possible the homogeneous irradiation of a predetermined volume of tissue.

Radium was also used in quantities of 1, 4 and 10 grammes in radium bombs, but because of the prohibitive cost of radium this use was restricted to a few centers.

Artificial radioactivity was discovered in 1934 and opened up an immense new field for medical and physiological research. It also provided new tools for radiotherapy.

Originally, only small quantities of radioactive isotopes were available, as they had to be produced in cyclotrons, but since the construction of nuclear reactors several hundred artificial radioactive isotopes are now available in ever increasing quantities. Of these, only a small number have been utilized in radiation therapy.

There are, first of all, those radioactive isotopes which can replace radium. Of these, Cobalt 60 is that most commonly used. Its radiation is similar to that of radium. It is very much cheaper to buy and it does not give off a gas as radium does, namely radon, so that it can be handled more easily. As I have mentioned before, Cobalt 60 is being used now in beam therapy units, and Manitoba will very soon have two of these units. Cobalt 60 has the disadvantage of a much shorter half life than radium—5.2 as compared to 1,620 years—so that the dose rate from radioactive Cobalt sources decreases somewhat from month to month. In the case of Cobalt beams, the unit has

to be replaced approximately every three years to run economically.

An isotope, used to replace radon seeds, is radioactive gold in the form of small grains. The advantage lies in the fact that these gold grains can be machined quite accurately and with the use of a gunlike introducer which was developed at the Royal Cancer Hospital, a number of grains can be discharged into a tumour from a magazine without changing the introducer. This instrument has been found particularly useful in implanting multiple small carcinomata in the bladder wall, and also in the palliative treatment of inoperable gastric carcinoma where they are inserted at laparotomy when radical operation has had to be abolished. The magazine is loaded with gold grains before activation in the nuclear reactor, so that exposure to personnel handling it is reduced to a minimum.

Another radium substitute is radioactive tantalum. It has a half life of 111 days and can be used in the form of a thin flexible wire which is silver-plated. This can easily be implanted into a tumour after exposure by surgery, and, because of its flexibility, can be withdrawn, as, for instance in cancer of the bladder per urethram. The isotopes, so far mentioned, all emit gamma rays, and it is these rays that are utilized in the treatment. Other isotopes only emit beta rays, and these have been utilized for radiation therapy when very superficial lesions only have to be irradiated. The two isotopes utilized for this are radio active phosphorus and strontium. The strontium has a long half life of 25 years and can be shaped into a shell, which can cover part of the eye and thus radiate such corneal lesions as may require radiotherapy, such as a chronic corneal ulceration, Mooren's ulcer, and vascularization of the cornea particularly before or after corneal graft. It appears, however, that this treatment is not entirely free from danger, as in a number of cases, revascularization has occurred, and very rarely radiation cataract of the lens has been noted. Phosphorus has been incorporated in a plastic material which can be shaped to any lesion as required and applied to it, so that very superficial rodent ulcers or hyperkeratoses can be treated with a minimum irradiation of normal tissue. I have already mentioned the use of radioactive gold grains. Radioactive gold emits both gamma and beta rays and it can also be utilized in a liquid form in the colloidal state and as such can be introduced into the pleura or the peritoneum in the case of malignant effusions particularly where no large masses are present, and in this type of case, it has brought about the cessation of secretion of fluid. This is not a curative procedure, as the short range beta rays which make up about 90% of the radiation cannot affect large masses, but it will make the

patient's life easier in that repeated tapping will no longer be necessary. In suitable cases, a good result can be expected from colloidal radioactive gold in approximately 40% of cases.

All the radioactive isotopes mentioned so far have been applied locally and I would finally like to turn to the use of radioactive isotopes administered internally. This represents a departure from radiotherapy as we have known it hitherto, because for the first time, we are trying to attack disease which may be spread throughout the body, without exposing the whole body indiscriminately to a dose of radiation.

The two isotopes used in this manner are Radioactive iodine and radioactive phosphorus. Radioactive iodine, like stable iodine, is taken up selectively by functioning thyroid tissue. Use has been made of this uptake, first of all to measure the activity of the thyroid gland, and iodine uptake studies nowadays represent one of the more accurate means of assessing thyroid function. It has also become an established form of treatment in the case of thyrotoxicosis, although the type of case in which it is best used is not yet universally agreed. It has been in clinical use for 16 years, without having caused serious sequelae, but some radiation effects, delayed even longer, may still be discovered, and therefore, perhaps, its use should be restricted in a benign condition like thyrotoxicosis, to patients who are beyond the reproductive period or in whom there are contra indications to the use of surgery, or in whom there is a recurrence of thyrotoxicosis following surgery. It is a simple form of treatment, and, as far as can be ascertained, the incidence of complications, such as thyroid crises, are exceedingly rare. In a certain proportion of cases, approximately 10%, myxoedema may be produced, which of course is easily controlled by the administration of thyroid. The concentration of iodine in functioning thyroid tissue has been utilized for the treatment of some carcinomas of the thyroid. It has been shown that in some cases, not only does the primary tumour in the thyroid take up iodine, but the metastases as well; this is more likely to occur with well differentiated tumours, and particularly those in which there is some colloidal formation. When all functioning thyroid tissue has been removed, the metastases, if they showed any inclination at all to take up radioactive iodine, become much more avid for this and therefore in treating carcinoma of the thyroid with radioactive iodine, it has been found best to ablate the thyroid in the first place by surgery or by a large dose of radioactive iodine. While, by and large, only the more differentiated carcinomas take up radioactive iodine, some undifferentiated carcinomas do so as well, and we have particularly noted two cases in which the metastases became progressively more anaplastic,

and, yet, these metastases continued to take up iodine. It is, therefore, worthwhile to study the iodine uptake in most cases of carcinoma of the thyroid where metastases are present. Unfortunately the proportion of cases exhibiting uptake high enough to make treatment feasible, is small and probably not more than 5% of all thyroid carcinomata can be dealt with successfully.

Of course, radioactive iodine can be utilized in a euthyroid patient where it is desirable to depress metabolic activity.

Radioactive phosphorus also, when administered by injection or by mouth, is not taken up indiscriminately throughout the body. It appears that there are two compartments, one composed of the bone marrow, liver and spleen and reticulo-endothelial system, and the other of the remaining soft tissues; the uptake of phosphorus would appear to be approximately nine times as high in the former as in the latter. Thus, obviously, the hemopoietic system can be irradiated selectively by internal administration of P^{32} . This in fact, has been utilized in the treatment of polycythaemia vera, in which radioactive phosphorus has become the standard treatment. In a great many patients, the administration of one or two doses of radioactive phosphorus of approximately 5 or 6 millicuries has been sufficient to obviate any further treatment for approximately 18 months to 2 years. This agent, too, has been used for approximately 14 or 15 years, and, as far as we can tell at present, no ill effects from it have been recorded. P^{32} is also administered internally to patients with chronic myelogenous and lymphatic leukemia, but the results here are not as good as those of external radiation therapy in most cases.

The subject of radiotherapy has now progressed to a point where one of our postulates can be fulfilled. It is possible to deliver anywhere in the body to predetermined volume a high dose of radiation without damaging normal intervening tissue excessively. Already it is clear that this progress alone will not bring about a dramatic improvement in the cure rate of deep seated cancer. The reason is not far to seek. Radiotherapy, like surgery, represents a local approach to malignant disease and can only deal with tumours locally. Once dissemination has occurred, in our present state of knowledge, external radiation hardly ever brings about a cure.

The field in which much further progress is needed is that of radiobiology. During the last 50 years, we have learned a great deal about the effects of radiation on living tissues. We have gained knowledge of the mechanism by which radiation affects tumour cells and the tumour bed, i.e. the normal tissues in close proximity of the tumour, and we have learned to appreciate the importance of the tumour bed reaction, but too

much is still unknown of the reason that makes some tumours more radio sensitive than others or the reason that will cause two cases to run such a different clinical course, although quite similar in clinical stage or histological appearance when first seen or what causes the growth of metastases 10, 15 or 30 years after the primary tumour has been eradicated.

A search is being made today for ways of influencing the biological response of tissues to radiation. If it were possible to find a "sensitizer" which would enhance the differential effect of radiation on tumour tissue as compared to the normal body structures, it might become possible to cure malignant disease after it had become disseminated. Gray in London has tried to increase oxygen tension in tissues in order to sensitize

tumours, while Mitchell in Cambridge has used synkavite, a vitamin K compound, for a similar purpose. Doubtlessly, this line of research will be explored further and will in time yield fruitful results.

Finally, it must be remembered that technological advance is only in part the answer to the problem. Radiotherapy is a clinical subject and the clinical appraisal of the patient and an intense study of the natural history of malignant disease are of equal or greater importance. These will enable us to evaluate the disease process and to choose the most suitable therapeutic agent for any particular patient afflicted with malignant disease. This clinical background alone will enable us to utilize to the full the new possibilities which progress in radiotherapy has offered us.

Medico-Legal

Memory and Some of Its Abnormalities*

A. T. Mathers, M.D., C.M., LL.D., F.R.C.P. (C.)

I must first ask your indulgence for some alterations I propose to make in the title of this presentation as it was originally announced. The chief item in that original announcement was "Abnormalities of Memory" and as a sub-title "Hypnosis" was mentioned. In thinking over the matter it seemed to me that before dealing with abnormalities of Memory something might well be said about Memory generally—its importance in life activities and the successive steps in the process of remembering. I am therefore making bold to redesignate this presentation as "Memory and some of its Abnormalities". As for Hypnosis, I propose to leave it out altogether.

The reason for this is that Hypnosis scarcely belongs among abnormalities of Memory. It is true that in the post hypnotic state there is no clear recollection of what transpired during the hypnotic state itself, but this is not the outstanding characteristic of Hypnosis—which is really a matter of extreme suggestibility in which the power of judgment is laid aside and proposals and propositions are accepted in the absence of any logical ground for their acceptance. Memory, or rather lack of memory, is involved, but, since it is not the sole component of the hypnotic phenomenon, this will be laid aside to give place to some things that should concern us more at this time.

When in "the dark backward and abysm of time" while Life pursued its slow and halting progression upward,—when did this thing or process we call memory begin to evidence itself? Did the primitive forms of life that have long since disappeared from the earth have the ability

to not only receive sensations but to store them away for future advantage and guidance? Did dinosaurs remember the pains and pleasures incident to their savage existence? Possibly not, else they would have continued to exist. Their failure to profit by experience brought about their downfall and elimination.

Do the lowly and relatively simple forms of Life that now exist remember? Observation of their simple structure, and the phenomena of their lives plainly suggest that for them there are no yesterdays. We know of phenomena in the lives of the less lowly forms that suggest some power of dim recollection. Does the female fish, her body heavy with the product of generation, indulge in the mad rush to reach the spawning ground where life first came to her? Do the migratory birds and insects recall the dangers of lingering too long in a region where inclemency cyclicly returns? Do they recall the soft and more luxuriant South? Does the bear, sleek and well fed, remember the peacefulness of his winter-long drowsiness? Not likely. Other factors, intricate and complex, both fateful and forceful, seem to initiate these insistent and unerring types of behaviour.

The animal, Man, appeared upon Earth at a time variously estimated from a few, to hundreds of thousands of years ago. His handiwork has survived as evidence of his existence for much longer than any actual discovered remains of his body. Did he recall to his advantage the pleasure or the misery or the vicissitudes of his life? It was no doubt "miserable, brutish and short". At first, little if any better off than the other creatures with which he contended in weald and wold, he must have slowly gained ascendancy, else they instead of him would have survived. And in this

*Read before the Medico-Legal Society, January, 1955.

wild and heartless competition there must have gradually and fitfully dawned a capacity to remember—first of all, things that meant Life or Death to him—later things not so fateful, but useful and appeasing—a sort of crude embroidery on Life—something to perhaps soften its harsh asperities.

Aeschylus has Prometheus, in mitigation of his bold defiance of the Gods, plead that he did much to transform Man's life to something better than mere brutishness. Says he, in speaking of primitive men.

"Let me tell you, not as taunting men
But teaching you, the intention of my gifts
How first beholding, they beheld in vain
And hearing, heard not, but like shapes in dreams
Mixed all things wildly down the Tedious Time
Nor knew to build a house against the Sun
With wicked sides, nor any woodwork knew
But lived like silly ants beneath the ground
In hollow caves unsunned. There came to them
No steadfast sign of winter, nor of spring
Flower perfumed, nor of summer full of fruit
But blindly and lawlessly they did all things
Until I taught them how the stars do rise
And set in mystery and devised for them
Number—the inducer of philosophies—
The synthesis of letters and besides
The Artificer of all things—Memory—
That sweet muse mother."

Man in his individual existence starting with conception lives through the successive stages of the life of the race from unicellular egg to the highly complicated finished product. The psychoanalysts theorize that even the foetus living in its quiet Nirvana-like state preserves some dim recollection of that semi-parasitic existence. Some go further and postulate a sort of racial memory citing as evidence the basic similarity of the myths and legends in which all racial groups attempt to explain the coming of Life and the conditions of its continued existence and progression.

Whenever it first appeared, Memory for us is one of the corner stones of Life. By reason of it we are able to register, retain and use not only the things experienced in our own lives for good or ill but the things that made Life what for the generations that preceded us and by whose successes and failures we profit. And in addition to this stark utilitarianism there are the innumerable pleasures that memory supplies. The recollection of past happiness and satisfaction go far to lighten the load when Life is too much with us. But memory has its terrors too—recall and persistent recollection of unhappy or evil happenings, no matter how far off and long ago they may have been, take the savour out of Life, and may render it a prolonged misery.

We are interested at this time in abnormalities of this important faculty—whether they be ex-

cesses, perversions or, what is more important, deficiencies. But before we deal directly with these let us consider just what the processes involved in memory are. The gross structural basis is a brain, one of a considerable degree of complexity and organization and furthermore, one that if healthy, can, up to a certain time of life, become more proficient in keeping with the extent of its use and training. The brain of an infant has potentialities which given fair play will eventuate, as life proceeds, in recognized capacity for remembering. As the vigor of life declines and involution comes more and more to the fore, this capacity along with others begins to fail, but in a certain fashion which will be mentioned presently.

The potentiality for intellectual development including memory resides in the brain cells, some billions of which make up what is called the cerebral cortex. This is the part of the brain by reason of which Man has attained his superiority over other form of life. One must not think of brain cells as empty spaces that gradually become filled. Each cell is in essence an electro-chemical unit in which all sorts of exceedingly complex happenings take place. Certain groups of these cells appear to subserve certain fairly specific functions. Some receive impressions from the outside world, some seem to initiate impulses that terminate in some kind of action related to the outside world and some, no doubt, correlate these mechanisms with previous experience. What actually goes on in these processes we do not know, but we have fairly good evidence that cellular activity is in reality chemical activity caused or accompanied by and resulting in some electrical changes. We must not think of the capacity to remember as localized to one area. It is a function of the entire cortex.

The first requirement in the process of memory is that an impression must be received, and it must have a certain potency. From time to time, indeed in all our waking hours, our senses bring us impressions, but many are so faint that they leave no trace. The impression that is to form the basis of a memory or recollection must, then, be a real one. A rather crude example will perhaps make the meaning clear. A light stroke over a piece of soft wax leaves no trace, an imprint with pressure does. Failure to make this necessary imprint on the concerned brain cells leaves no impression and the failure may be due to physical causes such as defect in any of the senses, the receiving mechanism, or to resistance on the part of cells themselves, the result of immaturity or disease, or to the intrusion of a variety of simultaneous sensations of preoccupations, as when in the midst of deep and complex thought we do not hear the nearby clock strike and have no recollection of it.

The next step is that the impression having been received must be retained. We have no idea of how this retention comes about. There evidently is some mysterious quiet shuffling about and reallocation of molecules that to date has defied detection by the most delicate instruments or procedures. With every sense impulse received, with every thought conceived, with every emotion felt, every act initiated this mysterious shuffling takes place. The remarkable thing is that some of it is but evanescent—a mere ripple, as it were, that moves and is gone leaving no trace of itself. But other shufflings leave something that is more or less permanent and under proper circumstances and because of this lasting rearrangement, the sensation, the thought or the feeling that started it may be brought back to consciousness, and we speak of this as remembering. Brain cells that are immature or improperly nourished, that are fatigued or crippled by disease will not retain the original impression. It seems probable too that a sudden violent emotional shock will at times interfere with retention, and this has important legal implications, as we shall see. Here again the psychoanalysts have a theory to offer. They aver that lack of remembrance due to failure of retention is not just a passive process like letting an object slip out of our fingers. Remembering some things would be uncomfortable or in some way distressing. They would have us believe that forgetting is often an escape from mental pain and Freud in his book, "The Psychopathology of Everyday Life," gives instances, more or less suggestive, that seem to give some support to this view. Ovid puts it thus—"We are slow to believe what, if believed, would hurt our feelings."

The next step in the business of remembering is Recall. That which has made an impression and which has been retained must be available for recall to consciousness. It must be, as it were, at hand and reachable at will much as one reaches into a cupboard for something previously placed there. In full remembrance the recall is distinct and complete. We can all think of instances when the recall is but partial. We may search about for some cue, some circumstance, that accompanied the original impression and by means of this association the recall may become complete.

Not only must we be able to recall—we must be able to recognize that which is recalled as being the same as was originally experienced.

The real faults in memory may be due to failure in initial impression, failure in retention, failure in recall or failure in recognition.

So much for the operation of the process of remembering. What are the abnormalities or deviations from the normal?

These abnormalities may be classified in several ways.

In one group we may place (a) amnesia or loss of memory, (b) hyperamnesia or excess of memory; (c) paramnesia or perversion of memory. Since amnesia is the phenomenon toward which our attention is chiefly directed this evening, we might perhaps dispose of the other two abnormalities mentioned in short order.

Occasionally in certain mental disorders we find that in relation to special points and special experiences, impressions arising from emotionally colored events are registered with more than usual intensity with the result that the patient has a vivid recollection of details. This is hyperamnesia, and has little or no medico-legal significance. Paramnesia—which means a falsification or distortion of memory—is most often noted among senile patients and some of those afflicted with mental disorder traceable to toxic or poisonous substances originating either within the body or without. What happens is that recent events make little or no impression and hence are not remembered. The patient then confabulates or tells a plausible story of what did happen. He falsifies but does not lie, in that the whole process is carried out without intention to mislead. The elderly patient with no recollection that he had not moved from hospital or home yesterday will tell a plausible story of being down town or elsewhere and will relate what he did. Akin to this is the quite unconscious falsification which old people indulge in when they relate in detail things that happened years ago or will repeat verbatim conversations that are said to have taken place many years gone by. As with hyperamnesia, this has little or no medico-legal significance, generally speaking.

However there are occasional instances most often in connection with overindulgence in alcohol in which impression or retention are disturbed. In the *Medico Legal Journal*, Vol. 20, part 2, 1952, Matheson—a psychiatrist evidently attached to Brixton Prison—tells of a man who during an extreme alcoholic spree, stabbed a man to death; relating his story straightforwardly but with entirely false coloring. His state when seen by the police, whom he himself called, was that of a man recovering from a heavy drinking bout. No motive was found, and the accused was a man of good character, never known to have shown violence toward anyone. The psychiatrist's opinion was that at the time he committed the homicide he was so drunk that he could have mistakenly believed, as he alleged, that he was stabbing a dummy when in fact he was stabbing a human being. The court accepted his plea of manslaughter instead of murder, as had been charged.

It is in connection with alleged amnesia or loss of memory that real difficulties arise. Amnesia may be true or it may be feigned in an effort to

escape responsibility for wrongful acts. It is important to distinguish the true from the false.

If amnesia or loss of memory or "blackout", to use a recently invented term, is put forward as a defence. There are with every emotion felt, every act initiated possibilities that must be explored.

These are (1) Hysteria

(2) Psychosis or severe mental disorder

(3) Alcoholism

(4) Low Blood Sugar

(5) Head Injury

(6) Epilepsy

(7) Malingering or defensive lying.

Hysteria as the name implies, being derived from the Greek word for "womb", was once believed to be a condition confined entirely to women. We know now, of course, that this is not so. It is the outcropping of a distinctive type of personality that may be evidenced in either male or female. Attention is likely to be drawn by the occurrence of dramatic episodes, but behind these and forming a background for them is a type of personality that is generally somewhat immature, is highly suggestible and much given to seeking attention, even if this is to be attained histrionically only. The individual with hysteria may appear with all sorts of apparent physical disorders. He may be blind, deaf, voiceless, paralyzed in some part of his body. But all these exist without evidence of actual physical disease of the part affected. To the individual these things are very real, although to others they savor of deception. Hysteria has at times been spoken of as "unconscious malingering"—a poor term since in malingering the person concerned is so clearly conscious of his position that he invents a means of escape from its implications, while in hysteria there is no conscious elaboration of a way out. People of hysterical makeup readily become involved in emotional conflicts in which personal drives and desires fall foul of existing circumstances. He or she may be full of hostility or resentment, may crave dependency and security and be generally unable to establish order and peace in his or her life. Anxiety is the result—and since it is the most unpleasant of all emotional states, the defences of the personality are mobilized against it. This is accomplished at a subconscious level—the patient does not deliberately seek an escape—it is provided for him. And one of the commonest forms of escape is amnesia—a forgetting of the whole business—awareness of the unpleasant features is blotted out. This mechanism is particularly liable to come into action in predisposed people faced with situations in which terror and shame and guilt or reluctance to face reality are present. The striking thing about memory loss is the patients indifference to it and the lack of perturbation displayed. He may revert to an earlier period

of life, behaving as a child, or may embark upon a fugue or flight in which he may travel long distances and even forget his own identity. The onset and termination of these amnesias is likely to be sudden. The differentiation from malingering is at times difficult and turns upon a biographical survey of the person's life. It is important to determine in occasional cases, whether the amnesia developed before or after the critical event.

Severe and devastating mental disorder is frequently attended by a blotting from the patient's mind of all recollection of events transpiring during the acme of the disorder. This is largely due to failure in impression and retention. The accompanying symptoms and signs serve to establish the nature of the amnesia, since it never is the sole evidence.

Alcohol in one of its many forms has since time immemorial been a source of forgetfulness, like the Waters of Lethe. This forgetfulness may be the chief object. Human beings, haunted by fears, sorrows or guilt, have sought for its solace but all have found that the peace of mind it gives is but shortlived. It is the great deceiver. With this use of alcohol we are but little concerned at present. There are certain people who because of peculiarity of personal organization respond to the ingestion of alcohol by becoming intoxicated with relatively small amounts, are likely to be aggressively violent and destructive under its influence and to have a complete or partial amnesia for the period of intoxication. Psychiatrists speak of this as pathological drunkenness, suggesting that there are some kinds of drunkenness that are not pathological. In the period of extreme overactivity, the person may commit crimes of violence, mostly of a senseless and unmotivated character, and when he recovers from his intoxication have no recollection of what happened. This amnesia is genuine and not something advanced in the hope of lessening culpability. The history of susceptibility to alcohol and the wild uncontrolled behaviour serve as distinguishing characteristics. Even if a court accepts the genuineness of the amnesia this does not always mean the acceptance of irresponsibility. In Anglo-Saxon countries generally the individual's responsibility is not lessened when he has voluntarily brought about a condition in which inhibitions are relaxed and primitive impulses to violence released. In most European countries the doctrine of partial responsibility is accepted.

Other substances that have effects similar to alcohol will at times produce amnesia for the period during which they are active. "The drowsy syrups of the East" and their more modern derivatives are among these. Some very modern synthetic drugs now utilized so extensively may at times have similar effects.

With increasing knowledge of the chemistry of the blood, we have become aware that when the amount of sugar in the blood drops to a low figure, definite clouding of consciousness with complete or partial amnesia may ensue and in the period of duration of this abnormality acts of violence may be committed. An overdose of Insulin in a person who is diabetic may bring about such a state, but it may also occur in persons who are not diabetic but whose kidneys allow too free an elimination of sugar or who are starved. Happily there is at hand a means of detecting temporary changes in brain activity that accompany this state of Hypoglycaemia or low blood sugar. The brain, for its normal activity, requires a constant supply of sugar brought to it by the blood. When the supply drops below a minimum, abnormal activity of brain cells ensues and conduct is altered. The electroencephalograph will detect evidence of this disturbed activity, which disappears when the supply of sugar returns to normal. Evidence of this kind was accepted as a defence in England within the past two years.

Head injury resulting in concussion will often produce a period of amnesia covering the actual time of the injury so that the patient has no recollection of the impact, but is also unable to recall events transpiring for an interval before the injury. We call this a retrograde amnesia—an amnesia extending backward in time. This amnesia may largely clear with the passage of time but rarely completely.

Then too we occasionally see an anterograde amnesia—one which extends forward in time. A person knocked out in a game, for example, may seem to recover, seemingly behave in a normal fashion but only some time later "comes to" so to speak and has no recollection of what happened.

Epilepsy is a much commoner condition than is generally believed. One reason for the belief that it is not common is the popular conception that convulsions are the only manifestation. There are many varieties of epilepsy, arising from different cause and with manifestations that differ widely. The one characteristic common to all epileptic phenomena is disturbance of consciousness and with disturbance of consciousness there is disturbance of recollection. This is not surprising.

But in some patients the convulsion is replaced or followed by marked clouding of consciousness in which the patient behaves in a more or less automatic and often violent manner. Hercules is alleged to have been subject to sudden attacks of blind rage in which he killed his best friends and his own children. Van Gogh, the painter, was subject to periods of irrational conduct of which he later had no recollection. In one he cut off one of his ears, wrapped it up and sent it to a lady friend. In his last attack he shot himself in the

abdomen. Of his behaviour which may be murderous, the patient, when he comes to himself, recalls nothing. He has an amnesia and it is genuine. This unremembered violence has two characteristics (1) its wild even ferocious character and (2) its motivelessness. There is no gain for the patient and if another person is attacked and injured or even slain, it is likely to be someone against whom the patient has no grievance, in fact it is likely to be the person nearest at hand.

In arriving at a conclusion as to the genuineness of this amnesia, one considers not only the facts just mentioned but also the history, revealing the occurrence of epileptic manifestations of one kind or another. Now there is an additional source of information and it is objective—that is, not modified by the personal impressions and accounts of the patient and his friends. This is the electroencephalogram or as it is commonly called, the E.E.G. This investigative procedure, stumbled upon almost accidentally and for a time considered an interesting but probably not significant affair, has been so developed that it is now of extreme value. To go into detail explaining it would take too much time. Briefly, the basis of it is the detection of abnormalities in the rhythm of electrical activity in the brain. The patterns of altered rhythm characteristic of epilepsy are pretty well known now. The test valuable and all as it is not absolutely conclusive since about 15% of people apparently normal show disturbances of rhythm and conversely about 15% of known epileptics show undisturbed rhythm. It should therefore be considered as part of a constellation upon which a diagnosis of epilepsy would be based.

The amnesia that is malingered and is really part of defensive lying is the source of the greatest difficulty.

Human beings, having been involved in unacceptable behaviour, resort to many subterfuges in an effort to free themselves from responsibility or if this is not entirely possible, to at least cast doubt upon it or soften the consequences in some degree. Denial of any recollection of having committed the act in question is a very common procedure. Resorted to in connection with acts that are no more than unseemly, ungracious or discourteous, it has no very serious implications—perhaps nothing more than some loss in credibility and trustworthiness.

When a serious delinquency or crime has been committed, duty has been neglected or testamentary capacity is in question, it is a different matter. Justice requires that the alleged amnesia or forgetfulness be most carefully scrutinized and appraised. As has been mentioned, there are conditions in which a genuine amnesia does occur. Acts may be committed in a state of clouded

consciousness and leave no recollection behind. In any case where amnesia is alleged, each of the conditions already mentioned must be considered and either eliminated or confirmed. The actual number of cases in which one or other of these causes can be confirmed is, I should say, small. The cases in which the amnesia is spurious are considerably larger in number. Conclusion that the alleged amnesia is feigned should not be reached until the conditions known to cause genuine amnesia have been eliminated. For such elimination there will be required:

- "1. a negative skull X-ray
2. an electroencephalogram which does not reflect either an epileptic or traumatic pattern
3. a negative examination of the nervous system
4. a life history which does not suggest hysteria
5. a picture inconsistent with alcoholic amnesia
6. a clinical examination negative for mental disease."

(Davidson).

The accused person must be interviewed a number of times. His first statement is of the utmost importance. It is in it that the truth is likely to be told. Charcot, the great French neurologist, spoke of the "period of contemplation", during which the person involved reviews his position, and is likely to elaborate and as he thinks, perfect his defenses. Dr. Matheson in his article in the *Medico Legal Journal* previously mentioned quotes the Rt. Hon. Sir Travers Humphry as saying "It is perfectly true that one of the most useful pieces of evidence which is ever given in a criminal case is the statement of the accused person before he or she has had time to think very much, consider their position or receive any advice, because in these circumstances you are likely to get the truth."

The first interview then is of great importance and if possible it should take place shortly after the act in question has been committed.

"If in repeated interviews, the account remains constant and if it is supported by known facts then it is likely to be genuine." (Matheson.)

"Generally a malingered amnesia is both patchy and self serving whereas a genuine amnesia usually extends to all areas of memory and is not confined

to facts that might hurt the defendant's case legally." (Davidson.)

The complete amnesia that is genuine is likely to include loss of personal identity as well as other things. This is not likely in feigned amnesia.

When the accused admits a dim and confused recollection; when he admits a partial recollection; when he remembers any incident that occurred between two incidents that are both forgotten, his statements should be regarded with the utmost suspicion.

With an experienced examiner much can be gained by close observation of the reactions of the accused during interview. It would be difficult to scientifically account for the "feel" of a case such as one with long experience achieves. There are subtle indications that are observable during the interview and which taken together are the foundation for this pretty trustworthy impression. How does the person interviewed sit on his chair? Does he displays restlessness of body or the small scope restlessness of the hands that betokens the uneasy mind? How does he answer questions—promptly and straightforwardly or with hesitation and shiftiness of gaze, as if he were being cautious and careful in his utterances? These are some of the things that might seem inconsequential only, often sway the balance when otherwise there may be uncertainty.

Sir, in perhaps too hurried a fashion I have dealt with the faculty of memory, on which so much of our life activities and experiences depend. It is in large part an unthought of and unacknowledged blessing. On occasion it may be the source of haunting sorrow and remorse. As with all human faculties it has its perversions and deficiencies. Some of these are disturbing and embarrassing in the life of the individual only. It is when they produce or are responsible for misconduct of social significance that they become of general importance. The appraisal of genuine disturbance of memory and the detection of alleged disturbance, advanced in mitigation of evils done, is a matter calling for the serious and sincere joint efforts of both Legal and Medical professions to the end that Justice may prevail.

Editorial

S. Valerub, M.D., M.R.C.P. (Lond.), F.R.C.P. (C.), F.A.C.P., Editor

Medico-Legal Society

Medicine has many relatives. Some are very close, others more distant. Anatomy, physiology, biochemistry, pathology, are practically members of the family. Biology, physics, psychology, although not living under the same roof, are close enough to be consulted in important family matters. Mathematics, sociology, philosophy and many others more distantly related drop in occasionally to exchange bits of gossip, bring gifts, lend or borrow.

Just where do we fit Law into this cosy family picture? The answer is not immediately apparent. Some of us will deny any kinship at all. Others, while conceding a distant relationship, would divest it of friendliness and cordiality. Many of us in answering this question would be biased by personal attitudes and experiences. We may harbor bitter memories of unpleasant experiences in court, when we were exposed to ridicule by a cross examining, quick witted, sharp tongued lawyer, or we may have more pleasant recollections of a rather vengeful nature, when this self-same self assured inquisitor became either totally inarticulate, or babblingly incoherent about his symptoms when relating his medical history in our office. We may recall, with a feeling of contemptuous superiority, a few amusing anecdotes about ambulance chasing lawyers, or by contrast, remember with humble gratitude some useful counsel received in an hour of need. Our personal experiences, thus, determine whether we regard them as friends or compare them with certain creatures of the sea. Yet we must not allow these personal relations between doctors and lawyers to obscure our view of the true relationship between Medicine and Law, for which we need the perspective of history as well as the panorama of contemporary life.

History tells us that Ancient Medicine drew its inspiration from religion and philosophy, and its guidance from Law. Basic sciences, the foundation of Modern Medicine, played hardly any part in the Medicine of ancient civilization, except that of Greece, where some Schools of Medicine attempted to base their therapies on current theories. Law, by contrast, played a guiding role. The Code of Hammurabi, the Lex Talionis, the Code of Justinian, all prescribed the standards of the practice of medicine, some of them going into such details as payment of medical fees, and punishment for negligence in treatment. The Laws of Moses governed the health of the community, enforcing

measures of hygiene and sanitation long before anything was known about the nature of disease.

Law fostered the growth of Medicine, and continued to regulate it through the ecclesiastic code of the Middle Ages, which tended to preserve the sacerdotal nature of Medicine, practiced according to set traditional pattern. The influence of the Law, even though less apparent, continues through the Modern Era. Thus, whether we deal with divine, common, natural or modern positive law, we always find an important allotment given to the problems of medical practice as far as they affect the individual and the community.

As we turn from history to look at the contemporary scene, we find that as doctors we are in contact with the Law every day of our lives. The Law regulates our rights and privileges in relation to our colleagues and our patients. It delimits the boundaries of professional secrecy and privileged communications. It penalizes malpractice, negligence, unauthorized operations, unjustified termination of pregnancy, sterilization, and other abuses. It calls on us for expert opinion in cases involving trauma and compensation, time and cause of death, drunkenness, drug addiction, testamentary capacity, insanity. It consults us on broader matters of policy with regard to problems of public health, sanitation, prescription and sale of narcotics, licensing of professional organizations, restriction on quackery and sale of nostrums. It seeks our advice where deep moral issues are involved, such as crime and responsibility and capital punishment. Thus, law pervades medicine as oxygen pervades the tissues.

The realization of the intimate relationship between Law and Medicine led to the founding in England in 1901 of a Medico Legal Society, in which members of the Bar and the Medical members met to discuss common problems and exchange views. The society publishes a quarterly known as the Medico-Legal Journal, which has been enjoying a growing popularity with members of both professions. Canada entered the arena rather late, for it was not until 1948 that the first Medico-Legal societies were founded in Vancouver and Winnipeg.

The local Medico-Legal Society has gradually grown in numbers and popularity. Its meetings are well attended, and the papers* presented are of a high caliber. It is felt by all that a field of liaison was created for the two professions which extended beyond the practical matters of mutual usefulness. For along with common interests and common techniques (gathering evidence and balancing probabilities) Medicine and Law have a

*A paper by Dr. A. T. Mathers, read before the Medico-Legal Society in January, 1955, is published in this issue.

stronger bond, that of common task of guiding, counselling and helping troubled humanity. Both deal with man and human misery, and as a result both develop along with a strong dose of cynicism a goodly quantity of pity, sympathy and the saving grace.

The Medico-Legal Society deserves well our support.

Manitoba's Medical Men XV. Health Insurance

Since earliest times doctors have believed that medical care is one of the basic human rights and to that end have given fully of their time and treasure and have not erected a "money barrier" between doctor and patient.

During the last generation the cost of medical care has greatly increased. This increased cost is partially due to the increase in the cost of drugs, surgical, food and other supplies. It is also due in part to the increased cost of building hospitals and the higher wages of the lay persons that work in the hospitals. As the cost of medical care increases, more and more of the medical dollar is used for hospitals, drugs and other expenses, and less and less of the medical dollar goes to the profession. In 1930 the doctor received 32 cents from the medical dollar and in 1950 he received only 28 cents of the medical dollar. Although his share is getting less with each decade the work that he does for no remuneration in the public wards of the hospitals continues as before. It can safely be said that the doctor is not responsible for the increased cost of medical care. In spite of this, the cost of medical care is increasing at an alarming rate and both lay and medical people are deeply concerned. Most governmental bodies have gone on record as favoring some form of health insurance, and the Canadian Medical Association also have gone on record as favoring some such plan. Doctors fear that in time health insurance may become state medicine.

The problem of health insurance is of much deeper significance than its increased cost to the people. In most of the countries where state medicine was introduced, the profession was not consulted. Of equally great importance is the fact that there is really not a free choice of doctor. Of the utmost importance is the fact that professional secrecy is not respected.

The Canadian Medical Association has had this whole problem under intense study for a long time. A report of their study to date was presented by the Chairman of the Committee on Economics, to the executive of the Manitoba Medical Association. His summary follows:

"The Canadian Medical Association has been interested officially in health insurance since 1930 and has favored some plan of insurance since

1934. In this respect health insurance is not used synonymously with State Medicine. The General Council of the Association states that well informed opinion today draws a sharp line between the terms and it is known that state medicine means complete regimentation of the medical services by the state, while health insurance means a pooling of financial resources by the prepayment of insurance premiums, whereby the cost of illness can be budgeted for in advance and the principle of free choice of doctor and patient prevail, with the least amount of interference of a third party between the physician and his patient. In 1944 when the Federal Government appeared ready to introduce a bill of health insurance, the Canadian Medical Association presented its points of view in a series of Principles to which it has since adhered. In 1949 the Association reiterated its attitude towards health insurance in its well known and fundamental statement of Policy which it has kept, together with the Principles, without change to the present time.

The General Council of the Association is aware of the changing conception of health insurance as it must evolve from both the state and the Profession. That there is a state of flux must follow from the wide spread use of voluntary pre-paid schemes sponsored across the nation by the profession in its several provincial divisions. A variety of problems of necessity must present themselves. The government will seek to understand what quality of professional service results from this type of practice, what costs will reach, what impact will be on the provision of good medical education, the facilities for caring for the sick and the effect on public health services. The profession will also have its problems of handling an increasing potential of patients, demands for new services, variations in income as many come to pay a fixed fee against the fluctuations of a sliding fee schedule, depending on the capacity of a patient to pay and the training of replacements in the profession with the eventual loss of the public ward.

Accordingly, the General Council has instructed the Economic Committee of the Association, composed of representatives of the ten provincial divisions, to draw up a consolidation of the two documents, viz., the Principles and Statements of Policy. This is to be done with a view to a clearer interpretation of what the Association attitude should be to the present attitude towards health insurance together with a wider interest towards the allied fields of Medical Education and research, public health services and the establishment of hospital and medical centres where they can achieve the greatest usefulness. Furthermore, the Association wishes to clarify its attitude in this consolidation of documents towards another

situation. In the event of a partnership being attained between the Government and the Profession in the general problem of the health of the people of the nation, the Association wishes to indicate what it feels should be the basis for a proper relationship between the parties involved, fair not only to the receiver of medical services but also to the provider of such services."

L. A. Sigurdson, M.D.

Letters to the Editor

Dear Editor:

In the October 1954 issue, Dr. James R. Mitchell presented a 3-year review of stillbirths, 1951 to 1953.

It is a known fact that two people studying the same series of cases can arrive at drastically opposed conclusions. This would be amusing if it were not also sometimes dangerous. On reading Dr. Mitchell's paper I apparently missed one short paragraph, and on studying the case histories I arrived at the conclusion that postmaturity is a definite factor in neonatal death statistics. It came as a surprise to me when I looked back on his article to see that he had decided, from the same cases, that postmaturity did not enter into the picture at all. Apparently, our pre-existing ideas and prejudices can color our interpretation of facts to a considerable degree, even when we attempt to apply scientific methods. The paragraph that I had missed was: "Contrary to popular opinion the incidence of intra-uterine death of unknown cause in the so-called postmature patient (i.e. over 42 weeks) who is healthy and does not suffer from toxemia, is very low."

What constitutes "death of unknown cause"? Dr. Mitchell puts 31 cases under this heading (not to mention 7 other cases under the heading "placental insufficiency (no toxemia)" and another 9 cases under "multiple pregnancy." Is this a cause of stillbirth? Or is the cause often anoxia; due, perhaps, to placental insufficiency, and this due, sometimes, to the fetus outgrowing its placental capacity?)

And what constitutes postmaturity? I submit that an arbitrary ruling of 42 weeks is unscientific and unsatisfactory, and that the postmature fetus is in danger of death from placental inadequacy at any moment. A woman who has had 7 perinatal deaths at term or in labor and who is allowed to go to full term in her 8th pregnancy, is probably "postmature" at "term" (i.e. 40 weeks) and she did in fact lose her 8th baby, induced at 40 weeks. This factor cannot be assessed in most cases; nevertheless these thoughts prompted me to analyze these 31 case histories presented, from the perhaps prejudiced point of view that 40 weeks is the limit of normal. Of these 31 cases, 17 were 40 weeks or

more. Authors may be quoted in support of the concept that postmaturity begins at 42 weeks or at 40 weeks, or that no definite time can be laid down—similarly that postmaturity does or does not contribute materially to perinatal mortality. I would like to mention only one—a very convincing article in the April 1954 issue of the *Journal of Obstetrics and Gynecology of the British Empire*.*

My purpose here is merely to point out that these same cases, studied by one who believes that postmaturity is a relative term and is definitely associated with perinatal deaths, seem to prove these facts; when studied by someone of the opposite point of view they may be said to bear out that opinion.

In summary, I believe these cases show that there is a relationship, and I further believe that the post-term pregnancy ought to be terminated in the interests of the fetus, even though one may be unable to fix a moment at which that particular placenta may become inadequate for that particular pregnancy. Expressed in other terms, the risk of induction at 40 weeks may be less than the risk of fetal death from placental inadequacy; and many obstetricians believe this to be often the case.

Yours sincerely,

M. Bruser, M.D.

*James Walker: "Fetal Anoxia." *J. Obs. & Gyn. Brit. Emp.*, 61, 2: 162.

Dear Editor:

Dr. Bruser has kindly permitted me to read his letter before its being printed in the Review so that my reply could appear at the same time.

Dr. Bruser queries my analysis of the causes of stillbirths at the Winnipeg General Hospital, 1951-53, particularly on the subject of postmaturity. The term "postmaturity" is an unfortunate one in the light of present knowledge of "placental insufficiency"—most of the current literature on the subject (which I will mention below) tends to stress the insufficiency of the placenta rather than a gestational period, which in many cases could be inaccurate. However, this literature is all very recent and most physicians still think of postmaturity per se as pregnancy advancing beyond 42 weeks. Postmaturity as a cause of stillbirths is, I agree, primarily fetal anoxia due to placental insufficiency.

I could only analyze the information available to me on the charts—in many cases this information was inadequate from the point of view of notes by the attending physician, description of the placenta, and autopsy report if available. Seldom does the attending physician describe the placenta in an abnormal case, yet this organ is the only means by which a fetus can survive the trials and tribulations of 40 or more weeks of gestation. Throwing

away a placenta that is not even described on the chart and then expecting the pathologist to give a cause of death on examining an apparently normal fetus, is locking the barn door after the horse is gone. Hence a long list of cases in my article with the cause unknown. Four cases of this group were over 42 weeks. In two cases there were contributing factors which indicated that it might not have been the prolonged gestation that contributed to fetal death, and in the other two there was just not enough information. Whether the other cases referred to in Dr. Bruser's letter were due to placental insufficiency is pure conjecture.

Those cases listed in the article as "placental insufficiency (No toxemia)" did have a description of the placenta and the final diagnosis reached by the physician and the pathologist (not myself) was that the placenta was just not able to adequately supply the fetus during the later weeks of pregnancy. Half of these cases are in the premature group, none go beyond 40 weeks.

Finally, the third group he refers to: "multiple pregnancy". The only contributing factor, as could be ascertained by a review of the charts, was that the pregnancy was not a single one, but in all other respects apparently normal. Eight of the ten cases in this group were premature.

As to the actual cause of death in these particular cases one can only surmise on the basis of information available. This is precisely what we of the Stillbirth and Neonatal Mortality Project, a federal government project going on simultaneously at the Winnipeg General Hospital and the St. Boniface Hospital, are attempting to investigate now. Placentas are examined and described—as much information as we can possibly attain on each and every pregnancy that results in a stillbirth or neonatal death is carefully analyzed, in order that we can eliminate these vague terms that we have been using in the past. If there was anoxia—why was there anoxia?

We all know that a fetus of a diabetic mother has a tendency to die before labor if pregnancy progresses beyond 37 to 38 weeks—apparently the placenta cannot carry the load. I agree with Dr. Bruser, that this can and does apparently happen in non diabetics who are quite healthy. How are we to recognize them before it is too late? It is ridiculous to consider induction in every woman who goes a few days past term—the possible complications alone of induction would raise, not lower, the mortality figures. Surely Dr. Bruser is not serious when he suggests this.

Physiologically, the placenta reaches its optimum efficiency by the 36th week of gestation. Thereafter its efficiency as an organ of transfer decreases. Calkins has shown that the fetus does not gain weight after the 39th week. Why does a pregnancy then tend to last 40 or more weeks?

If we knew what causes a woman to go into labour, perhaps we would know the answer. The incidence of pregnancy progressing beyond 42 weeks varies between 5-7%. Fetal and neonatal mortality (as studied by Clifford in Boston) does **not** increase in the multiparae who go beyond 42 weeks, but it **does** increase considerably in primiparae (especially the older primiparae) beyond 42 weeks. Clifford estimates 10% of primiparae lose their babies (the majority before labour). This incidence does not begin to rise until the 42nd week is passed. Walker of Aberdeen, whom Dr. Bruser refers to, has contributed a series of articles to the literature on this problem. In conjunction with Turnbull he has shown that fetal hemoglobin tends to rise after 40 weeks, a compensatory mechanism for lowered oxygen saturation of fetal blood returning from the placenta.

Therefore, Dr. Bruser is probably correct in suggesting that 40 weeks is the limit of normal on physiological grounds, but does that mean every woman who goes a day beyond 40 weeks should be rushed into hospital and induced? One thing I believe we all tell our patients is that the expected date is only a relative date and not to expect the pregnancy to terminate by that date. Certainly we emphasize this to primiparae, who, in my experience, frequently go beyond their dates and then deliver happily. There must be a good safety factor present in the majority of placentas to allow this. To become alarmed the moment a healthy woman, who is not toxic, is well nourished with a good hemoglobin and a normal developing fetus, goes beyond 40 weeks is asking for unnecessary trouble. If one is **sure** of the development of gestation, and pregnancy has progressed beyond 42 weeks, then I believe surgical (not medical) induction might be considered in view of the possibility of the placenta not carrying the load. Nolle in Stuttgart (1950) began inducing labour in 6.8% of his series of 2,300 cases beyond term. His stillbirth rate was 11.1%, over half of which he believed due to the induction. The remaining cases were allowed to progress until spontaneous labour began. The stillbirth rate was then 0.7%.

Perhaps, with further knowledge, we will be able shortly to recognize properly the dangerous pregnancy where the placenta is not able to carry the load to term despite other factors being normal, but until then I submit we continue to use 42 weeks as the top limit of our safety factor before we become concerned, and continue our study on those cases in which the placenta matures long before the average.

Very truly yours,

James R. Mitchell, M.D.

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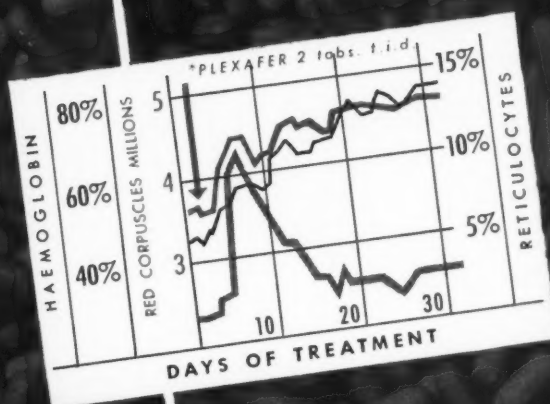
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Social News

Reported by K. Borthwick-Leslie, M.D.

Sunday p.m., March 13th,

Moana Hotel, Honolulu, Hawaii.

To ye, new editor, my first gesture of insubordination! You are being thoroughly cursed, may the shades of all literary, scientific and social miseries follow you and burn your conscience, yea forevermore, for bullying — yes, ladies and gentlemen, bullying me into spending a glorious Sunday in my room digging up "Snoopers" notes, while my running mates, Ida and Helen, bask and also burn on the beach under the greatest Banyan tree in the world.

Oh! well, since my first order from the C.O. is to publish Athol Gordon's toast to the ladies and Doris Gowron's (Mrs. Tony) response given I will have to be one of those ladies and get on with my job as affably as possible.

On February 12th, as you may recall, the Practitioners' Association held the Annual Dinner and Dance. Completely successful and entertaining as always, particularly Dr. Athol Gordon, in his own inimitable manner tracing the advent of "Women" from that first excision of rib through the green apple episode and the downfall of poor Adam, on to maternity, paternity, fraternity, practically up to fraternization between all nations, sects, etc., even in the time of Helen of Troy, Cleopatra and on to the modern generations. From my window here, I am sure Athol would be able to dig up the replica of "The barge she sat in like a burnished throne, the poop of beaten gold (\$1.50 for 15 minutes of Outrigger Canoe), purple the sails and so perfumed even the winds were lovesick (?). The oars of silver which to the flutes kept stroke, making the water which they beat to flow faster as amorous as their stroke." So on ad infinitum — which all boils down to that fact that Athol keenly appreciates us gals and makes no effort to disguise the fact. Cleverly and well done, Athol, much appreciated by all.

In her response Doris Gowron, yea, also in her own charming manner, expressed our thanks for Athol's "gilding of the lily." Her brief summary of the trials of a Doctor's wife was reminiscent of days long gone for a number present, but Tony and confreres are, for sure, able to take a bow!

I cannot resist some comment re Athol though, in all the years we have studied together, I definitely knew and appreciated his prowess as a musician, student of the classics and latterly soldier, historian, artist, etc., but until Carlyle Allison in his "Corner Cupboard" came through with the "Athol Gordon Week" I never suspected the Sunday School Leader phase and the impact of his personality on the youth of that day. Hi, pal, the post brim of my hat is tilted! So much for the Athol Gordon week.

Thank you, Dr. Purdie, for your nice comments on my feeble efforts, also for chastising me for lack of "snooping" re the Old Boys and Gals I presume. You may enjoy my later comments and after my sojourn in B.C. should have more news. I do find though that particularly in my Stork Dept. the younger fry are more active! Congratulations to you and your son. Yes, I believe you are a "First" — in that you, in the Council of C. P. & S., represent the Profession in District 7, Brandon, while your son, Dr. F. J. E. Purdie, Griswold, represents Electoral District 6, both for the Four-year term — 1954-1958.

What goes on at home, I do not know, but hope someone is going through those papers for me, or all will be lost next month.

We — in case you do not miss us, that is Drs. Ida Armstrong, Helen Marlatt and myself — finally did get ourselves packed and aboard the T.C.A. March 1. Uneventful flight to Vancouver, to be met, wine and dined by my big brother between flights. Vancouver that day looked a bit like Manitoba — golf courses covered with snow, much to everyone's horror. On to San Francisco, most interesting, but bumpy, because of storms, fog and flitting about from one lane to another, dodging other planes, plugged ears, muscle cramps but no necessity for those intriguing cups reading "After use please fold over top before discarding." Enjoyed the longest, strongest Manhattan I have ever tasted, courtesy of the airline. You should have seen Helen attacking a decanter full of Scotch! She asked for Scotch and water and she surely got it — neat. Everyone aboard felt much better for the therapeutic treatment though.

San Francisco to me is fabulous — ouch, those shops, bang goes next month's income, can foresee meat balls far into the future.

The Lurline S.S. crossing was fun. Very cold, wet and rough for 36 hours, but again no meals missed — unfortunately we found when trying to gather the "Ah Poos" (abdomen to you) into our clothes. Five days crossing and loving every minute, but Honolulu, oh! boy. The natives think the weather terrible, windy, wet, etc., but to us, like May and June. The gals, being athletic, have taken to Surf Riding, Golf, etc., but I find that extra hinge joint in the Humerus disconcerting, so inhale my iodized salt water in small quantities close to shore.

Social News (Continued)

We arrived too late for the Drs. Fahrni (Sr.) and Abbott sojourn, but have contacted Bunny and Mrs. Munroe — who look wonderful — in spite of the fact that they have just become Grandma and Grandpa.

They have a Jap houseboy who calls Bunny "Duck" and amused everyone by asking one day: "Duck, how old you? Your little wife Japanee and much Young?"

Have it on good authority by local snoopers that before they left, the Fahrnis had one of the wettest cocktail parties on record.

The rains came, not in drops but in waterfalls like turning on the taps. Guests arrived carrying footwear, clutching skirts and trousers near the

midriff. Same guests going home as wet internally as externally, not so concerned about appearance.

More anon — We have reached the stage of relaxation that this A.M. one room-mate remarked: Oh! dear, do I wash these undies or just turn 'em over and wear them tomorrow?

Dr. and Mrs. I. C. Peever of Fort Churchill, Man., are happy to announce the arrival of Douglas Irvin, brother for Sandra, on February 22, 1955.

Dr. E. D. Hudson (Man. '87), an ardent curler until two years ago, was made a Life Member of the Hamiota Curling Club. He still enjoys watching the glorious game and had the honor of throwing the first rock at the 60th Anniversary of the club.

Victorian Order of Nurses 1954

The Winnipeg branch, Victorian Order of Nurses serves the cities of Winnipeg and St. Boniface and the following municipalities:

East, West, North and Old Kildonan.

East and West St. Paul, St. Vital.

Fort Garry, Tuxedo, St. James and Brooklands.

Nineteen nurses cared for 3,116 patients in 1954, making a total of 38,903 visits.

This was an increase of 4,755 visits over those made in the previous year. The increase in work was accomplished by the same number of nurses by provision of quicker transportation. This was done by renting U-drive cars. Transportation costs were of course increased, but the nurses were nursing rather than waiting for street cars.

May we remind you that Victorian Order service is available to anyone who needs nursing care in their home. The average visit costs \$2.00 and those who can afford it pay this amount. The fee is scaled downward or free care is given when indicated. The nurse who visits the home discusses the fee with the family and together they agree on a suitable fee. Factors which are considered if a reduction in fee is requested are:

- (a) Family income
- (b) Size of Family
- (c) Probable length of illness.

Physicians' Art Salon

The Physicians' Art Salon Committee invites any Canadian physician or medical undergraduate to enter his work in the 1955 Salon to be held in Toronto's Royal York Hotel from June 20 - 24. This will mark the 11th year for this popular art and photographic feature of the annual C.M.A. Convention. It is sponsored by Frank W. Horner Limited, Montreal.

Conditions of Entry

The Salon structure will undergo few changes. As in previous years, entries will be accepted in three sections.

1. Fine Art
2. Monochrome Photography
3. Colour Photography

The Fine Art section is further subdivided into Traditional, Contemporary, and Portrait categories. There is no restriction on medium, — oil, tempera, gouache, water colour, charcoal, pencil, or dry brush is acceptable in each.

However, each exhibitor is limited to three entries in Fine Art and Colour Transparencies, but in Monochrome Photography four prints are permitted. And any exhibitor may enter up to the limit in one or more sections.

There is no charge, — all costs, including transportation of entries to and from Toronto, will be borne by Horner.

Judging and Awards

All acceptable entries will be displayed prominently in the Salon and then judged for awards by a competent jury to be selected by the Art Salon Committee.

To Obtain Entry Form

Any physician or medical undergraduate interested in submitting work may obtain an entry form with details by writing the sponsor at P.O. Box 959, Montreal. A short note or post card will do. The entry form contains complete instructions on how to prepare and ship your entries.

Art Salon Calendar

A novel feature of the Salon, the Physicians' Art Salon Calendar, will again be prepared by Frank W. Horner Limited. The Calendar reproduces award-winning work in full colour and is distributed to all physicians in Canada, with the compliments of the Company.

Association Page

Reported by M. T. Macfarland, M.D.

North of 53 District Medical Society

A meeting of the North of 53 District Medical Society was held at St. Anthony's Hospital, The Pas, Manitoba, on the afternoon of February 11th, 1955.

Present were Doctors: C. S. Crawford (Chairman), The Pas; J. Leicester (Secretary), The Pas; A. L. Jacobs, The Pas; R. F. Yule, The Pas; P. G. Premachuk, The Pas; N. Stephansson, Flin Flon; E. Redpath, Flin Flon; S. Carey, Clearwater Lake; J. Silinsky, Clearwater Lake; J. B. Johnston, Snow Lake; R. T. Ross, Winnipeg; L. R. Coke, Winnipeg; E. W. Pickard, Winnipeg.

Dr. N. Stephansson was elected as president, and Dr. E. Redpath as secretary of the society for the forthcoming year. Dr. P. Johnston was elected as the District Representative to the Manitoba Medical Association for the next year.

A very instructive and interesting scientific session was held. Dr. R. T. Ross presented a paper on Fits and Faints, bringing in the newer concepts as to the origin of abnormal electrical discharges in epileptics. Dr. L. R. Coke discussed the widening scope of cardio-vascular surgery and its indication in congenital and acquired heart disease. Dr. E. W. Pickard outlined the surgical management of burns and the treatment of skin defects.

Following a social hour at the home of Dr. C. S. Crawford an enjoyable dinner was provided by the Sisters of Charity at St. Anthony's Hospital.

The visiting doctors attended the Mad Trappers Rendezvous with some of the local medicos.

There were ward rounds on the morning of Feb. 12, 1955 where interesting cases were discussed. Two cases of acute coronary thrombosis were seen by Dr. L. Coke. Three neurological cases were presented for Dr. R. T. Ross to examine and discuss. The medical society found the meeting very enjoyable and instructive.

J. Leicester, Secretary.

Brandon and District Medical Association

The following is a Report of a Meeting of the Brandon and District Medical Association held in Brandon on the 17th of February, 1955.

The meeting took the form of Medical Rounds with the presentation of several cases. Mr. A. McTaggart, Administrator of the Brandon General Hospital very kindly arranged for space in the former Medical Ward at the Hospital and there was nothing lacking in equipment from blackboards to ash trays. Thank you, Mr. McTaggart. We hope we can use your facilities again.

The following cases were presented from 2.00 p.m. to 5.00 p.m. after a very delightful luncheon at the Brandon General Hospital

Peripheral Vascular Disease — 3 cases:

Occlusive Arterial Disease.....	Dr. R. P. Cromarty
Post Phlebitic Sequelae.....	Dr. H. S. Evans
Edema of the Legs.....	Dr. F. J. E. Purdie
Myelogenous Leukemia.....	Dr. R. F. Myers
Empyema in an Infant.....	Dr. J. B. Baker
Acute Glomerulonephritis.....	Dr. J. B. Baker

Dr. Reed Taylor gave an interesting account of office procedures in Prostatism.

Drs. N. Merkeley, Paul Green, Reed Taylor commented on the various cases presented and the Meeting was very grateful for their help and timely comments. Thank you, gentlemen. We hope we may call on your services again.

At a Business Meeting following the Clinical Session the following officers were elected for a period to extend to May, 1956 to coincide with the Manitoba Medical Association Elections and to save having another election in May, 1955.

President — Dr. F. J. E. Purdie..... Brandon
Vice-President — Dr. G. T. McNeill..... Carberry
Secretary-Treasurer — Dr. V. Sharpe..... Brandon

Dinner was held at the Prince Edward Hotel with 47 guests present. Following dinner, Mr. Jack Cunnings, Winnipeg brought us up to date on Rehabilitation in the province and on the recently established pension for the totally disabled. Mr. Cunnings was introduced by Mr. A. McTaggart, Administrator of the Brandon General Hospital.

The meeting adjourned about 10.00 p.m.

V. J. H. Sharpe.

General Practitioners

General Practitioners' Association of Manitoba
In Affiliation with the Manitoba Medical Association

The series of post-graduate lectures sponsored by the General Practitioners' Association of Manitoba continues with unabated interest. The very practical approach, so suitable for G.P.'s has been carried on by each lecturer.

Dr. Burrell's talk on Peripheral Vascular Disease emphasized the following important points:

Differentiation of post-phlebitic ulcer and varicose ulcer.

The classical picture of Raynaud's Disease and the much more common Raynaud's phenomenon. The former occurs only in women, it is usually bilateral, may be in any of four limbs and shows

characteristic biphasic color changes. The pulses of the affected limbs are normal.

Raynaud's phenomenon may occur in:

a. Pneumatic hammer disease (and therefore in males).

b. 10% of people with atheroma.

c. Post-traumatic dystrophies.

d. Post polio.

e. Ergot poisoning (usually patients with migraine).

f. 40% of people with Buerger's Disease may have some arm involvement and this may take the form of Raynaud's phenomenon.

A condition of patchy cyanosis known as Livido Reticularis was described. It often occurs in the newborn and is carried over into adult life. Sympathectomy will help but will not improve the appearance.

Acrocyanosis which occurs in women is characterized by limbs which are cold, sweaty and blue and can be cured by sympathectomy.

Frost bite, Immersion Foot and Trench Foot are now best treated by rapid defrosting, anti-coagulants (Heparin 75 mg. to 100 mg. o.h. iv) antispasmodics, (papaverine, nicotinic acid. alcohol). Sympathectomy as an emergency operation may be done if the above measures are not sufficient.

Arteriosclerosis, Arterial Emboli and Thrombosis.

Arterial emboli are often missed in the first few days. A routine examination of the radial pulses

of middle aged and elderly people should be done especially if there is a history of heart trouble. Embolectomy is best done in the first 12 hours but may be done up to 24 hours.

Some splendid films were shown illustrating the pinching-off of vessels as shown by arteriograms of limbs. This narrowing characteristically occurs where branches are given off. The sclerosis is patchy in contradistinction to the relatively innocuous Monckberg's Sclerosis where often the whole vessel can be seen on the plane X-ray film.

Intermittent Claudication or pain brought on by effort when occurring in the legs usually means that the vascular disease process is fairly high (above the Popliteal) and is important. Absence of the dorsalis pedis is only important if there is also coldness of the affected side.

Treatment may be briefly summarized as follows:

1. Limitation of animal fat, cholesterol.
2. Anticoagulants—long term use.
3. No smoking. It is important to emphasize that smoking definitely causes vasospasm and to give the best possible chance to the collateral circulation it should be absolutely forbidden.
4. Sympathectomy must be at a high level so as to be sure that vessels above the affected segment and taking part in the collateral circulation will be included.
5. Avoidance of cold.

A. G. Henderson.

Medical Events Calendar

Winnipeg Medical Society

Monthly Meeting—Third Friday, 8:30 p.m.

Winnipeg General Hospital

Surgical Case Presentation, Tuesday, 1:00 p.m.

Tumor clinic Wednesday, 9:00 a.m.

Ward rounds, Wednesday, (W3 Balcony), 1:00 p.m.

Clinical luncheons, first and third Thursday, 12:30 p.m.

Psychiatric clinic, Thursday, 10:30 a.m.

Neurological clinics, Friday, 1:00 p.m.

Outpatients ward rounds, Saturday, 11:00 a.m.

St. Boniface Hospital

Clinical mornings, Wednesday from 9:00 to 12:00 a.m.

Clinical luncheons, second Thursday, 12:30 p.m.

Tumor clinic, Friday, 11:00 a.m.

Victoria Hospital

Clinical luncheons, third Friday, 12:30 p.m.

Deer Lodge Hospital

Clinical luncheons, first Monday, 12:15 p.m.

Clinical Pathological conferences, Tuesday, 1:00 p.m.

Ward rounds, Thursday, 11:00 a.m.

Chest conferences, third and fourth Thursday, 12:45 p.m.

Misericordia General Hospital

Clinical luncheons, second Tuesday, 12:30 p.m.

Grace Hospital

Clinical luncheons, third Tuesday, 12:30 p.m.

Children's Hospital

Ward rounds, Thursday, 11 a.m.

Clinical luncheon, Friday, 12:30 p.m.

Congenital heart disease clinic, third Tuesday, 12:00 p.m.

Municipal Hospitals

Clinical luncheons, fourth Friday, 12:30 p.m.

College of Physicians and Surgeons of Manitoba

Council Meeting (Continued)

October 16, 1954.

G. Report of Representatives to the Cancer Institute.

Under the provisions of the incorporating Act the President and Registrar of this College are ex-officio members of the Institute. While half of the medical representation from this body changes yearly, the registrar has been a member of the Board for the past six years. During that period a medical member representing the University of Manitoba and another medical member appointed Chairman of the Medical Advisory Committee with the three representatives from the Manitoba Medical Association, each of whom is appointed for a three year term, one to retire each year, constituted the Medical Advisory Board. When, following considerable negotiations, diagnostic facilities were made available for indigents or near-indigents from rural Manitoba, a hospital Committee was set up in each of the participating hospitals, the Medical Advisory Committee fell into comparative disuse. In addition, although considerable ground-work was done by individuals and committees, and policies arrived at without prior reference to the Board, members assumed responsibility for all actions of each and every Committee by participation in the "omnibus resolution" proposed at the end of each annual meeting.

At a meeting of the whole Board in November, 1953, some remarks were made by your registrar, and various information was sought. Confirmation was sent by letter on November 25th, but no reply has been received at this date.

"Although the copy of the Report for 1952-53 arrived on November 19th, I did not peruse it until a short time before the meeting, hence I had no prepared copy of the remarks which I made at Monday night's meeting.

Attention was called to the statement on page 9 under heading (b) that diagnostic facilities are not adequate in rural Manitoba. The fact that there has been a continuous increase in the settlement of medical men in rural Manitoba, and that the biopsy and diagnostic facilities are available, in the former for all rural residents and in the latter for needy persons, leads me to suggest that there is not now the marked contrast between rural and urban facilities which may have existed several years ago.

On page 19 under "Professional Education", I would be interested to learn what measures were carried out by the Institute during the year, in addition to the efforts for "Lay Cancer Education" outlined on page 18.

It was noted that on page 20 the number of biopsies received was from rural physicians only, that the service was one to assist in the diagnosis rather than purporting to be diagnosis by mail (reference Winnipeg Tribune, March 14, 1953, 'How Manitoba is Fighting Cancer by letter'). On page 23 it was felt that the total of new cases treated by X-ray would indicate that both rural and urban cases were included.

The figures which I used from the Annual Report in relation to the Financial Report which was presented in May were as follows: 109 patients referred to the diagnostic services at a total cost of \$49,899.40, would give an average figure of \$457.00 per patient.

The 500 new cancer cases treated by X-ray divided into \$128,570.72 would give an average cost of \$257.00, and of the 1991 new cases reported, since only rural cases are paid for, the average cost would be in the vicinity of \$290.00.

The fallacy of arriving at figures in such manner was also outlined at the meeting to make the point for other members of how little is known of the day-to-day operation of the Institute.

The questions concerning the proposed revision of the Board establishment, the possibility of a diagnostic service for Brandon, were to clarify matters on which there has been previous discussion and queries from my confreres in the Province.

Dr.'s application for basic registration, apart altogether from his qualifications as a specialist in his particular field, has not yet been formally dealt with by the Registration Committee of the College, although the necessary information is on hand. P.S. Since dictating above, Dr.'s application has been approved."

The following report was presented at the Annual Business Session of the Manitoba Medical Association on Wednesday, October 13, 1954, and with the permission of the author, the Association, and this Council is being included in this report: (Report follows)

Recommendations:

That during the next year a study be made by your two representatives, independently or in co-operation with the Cancer Committee of the Manitoba Medical Association, of the Incorporating Act, the by-laws of the Institute, the status of medical employees in relation to the Executive Director, and the present status of the Cancer Diagnostic Clinics. The relation of the Institute to the Community Chest of Greater Winnipeg, also to the Canadian Cancer Society, and National Cancer Institute. It is always a source of doubtful satisfaction to learn that all the other Canadian Provinces are out of step except Manitoba. Contact

was made with the College Solicitor to ascertain whether the duties of either President or Registrar might be delegated to another member of this Council, but it is the verbal opinion that such is not possible.

Respectfully submitted,

M. T. Macfarland, M.D., C.M.

Motion: "THAT the report of the Representatives to the Cancer Institute be adopted." Carried.

Dr. C. H. A. Walton stated that the Registrar should be congratulated on his report since it is exceedingly important that the Council know the difficulties which have been arising. It seems that from the point of view of Council the recommendation of the Registrar should be dealt with. If this Council requested it, the Institute might ask the Legislature to amend the Cancer Act to appoint two representatives instead of officers. The difficulties are that the President often lives a long distance away and it is difficult for him to get into the meeting which might be frequent and for which there is no financial support, and that the Registrar is often put to considerable embarrassment as he is a representative of all doctors in the Province which makes it difficult to take a stand which might not be agreed to unanimously by the doctors of the Province. He thought that if the Cancer Act could be amended, the Council would feel happier to appoint two members. The failure of the Executive Officer of the Institute to acknowledge or reply to a former letter from the Registrar is lamentable but quite characteristic. He stated that as a member of the Advisory Board he would like a copy of the Registrar's letter to show what has been done 11 months ago.

Dr. C. E. Corrigan stated that he had served on the Advisory Board for a long time both before and since the war and it had always been his impression that the Board acted as a rubber stamp, and it was his feeling that he wanted to get off the Board. He said that the C.P. & S. has certain duties to perform, and suggested that the discussion might be deferred because Dr. M. R. MacCharles was not present at this Council meeting, and he was Chairman of the Advisory Board.

Dr. Walton stated that Council was not prepared to deal with such an involved matter and suggested that the minutes of this meeting would contain the Registrar's report in detail for consideration by the Council. The Advisory Board may produce changes which would not make it necessary for this Council to act. This is an educational introduction and perhaps the Council may have to act in the future.

**H. Representatives to the Liaison Committee —
M.M.A. and C.P. & S.**

The Registrar reported there had been no meetings held since the May meeting of Council.

I. Representative to the Canadian Arthritis and Rheumatism Society — (Manitoba Division).

The Registrar advised he had received no report from the previous representative to the Canadian Arthritis and Rheumatism Society — Manitoba Division.

Dr. A. R. Birt questioned the advisability of continuing with a representative to this Society since it has been linked up with the Community Chest, and wondered whether Council should be represented on any such bodies.

**6. Election of Officers and
Standing Committees.**

Officers

A. President:

Motion: "THAT Dr. C. H. A. Walton be appointed President." Carried.

Dr. T. W. Shaw vacated the Chair in favour of the newly-elected President, Dr. C. H. A. Walton.

B. Vice-President:

Motion: "THAT Dr. Percy Johnson be appointed Vice-President." Carried.

C. Registrar:

Motion: "THAT Dr. M. T. Macfarland be appointed Registrar." Carried.

D. Treasurer:

The Chairman stated that the former Treasurer, Dr. T. H. Williams was no longer a member of Council but referred to Section 17 of the Medical Act which reads in part: "The council shall annually appoint from amongst themselves a president and a vice-president, and from the members of the college a registrar, a treasurer and such other officers as may from time to time be necessary for the working of this Act, —"

Motion: "THAT Dr. T. H. Williams be appointed Treasurer." Carried.

**Nomination Committee to Strike
Standing Committees.**

The President appointed Doctors Ed. Johnson, C. E. Corrigan, and F. K. Purdie, as a Committee to Strike Standing Committees.

Standing Committees

A. Registration Committee:

Dr. C. E. Corrigan, Chairman
Dr. A. R. Birt
Dr. R. E. Beamish

B. Education Committee:

Dr. T. W. Shaw, Chairman
Dr. A. L. Paine
Dr. A. E. Childe

C. Finance Committee:

Dr. F. K. Purdie, Chairman
Dr. T. H. Williams
Dr. M. R. MacCharles

D. Legislative Committee:

Dr. J. M. Kilgour, Chairman
Dr. A. L. Paine
Dr. S. S. Toni
Dr. F. J. E. Purdie
Dr. A. R. Birt

E. Discipline Committee:

Dr. Ed. Johnson, Chairman
Dr. R. E. Dicks
Dr. F. P. Doyle
Dr. R. E. Beamish
Dr. F. K. Purdie

F. Executive Committee:

Dr. C. B. Stewart, Chairman
Dr. G. H. Hamlin
Dr. Ed. Johnson
Dr. M. R. MacCharles
Dr. F. P. Doyle

G. Taxing Committee:

Dr. Percy Johnson, Chairman
Dr. S. S. Toni
Dr. J. M. Kilgour

Representative to Library Committee:

Dr. A. E. Childe

Motion: "THAT the appointment of Standing Committees be accepted." Carried.

Election of Special Committees**A. Representatives to the Manitoba Medical Association Executive:**

Motion: "THAT our representatives to the Manitoba Medical Association Executive be Dr. Ed. Johnson and Dr. C. B. Stewart." Carried.

B. Representatives to the Committee of Fifteen:

Motion: "THAT our representatives to the Committee of Fifteen be Dr. J. M. Kilgour, Dr. A. L. Paine, and Dr. A. R. Birt." Carried.

C. Representative to the Committee of Selection in Medicine:

Motion: "THAT our representative to the Committee of Selection in Medicine be Dr. R. E. Beamish." Carried.

D. Representatives to the Medical Council of Canada.

Motion: "THAT our representatives to the Medical Council of Canada for the current four-year term of the Medical Council of Canada be Dr. C. E. Corrigan and Dr. C. H. A. Walton." Carried.

E. Representative to the University Senate:

Motion: "THAT our representative to the University Senate be Dr. C. H. A. Walton." Carried.

F. Representatives to the Liaison Committee — M.M.A. and C.P. & S.:

Motion: "THAT our representatives to the Liaison Committee — M.M.A. & C.P. & S. be Dr. Ed. Johnson, Dr. C. B. Stewart, and Dr. F. K. Purdie." Carried.

G. Representatives to the Specialist Committee:

Motion: "THAT our representatives to the Specialist Committee be Dr. C. H. A. Walton as Chairman, and Dr. F. K. Purdie." Carried.

Appointment of Auditors:

It was agreed to leave the appointment of auditors until the May meeting of Council.

Appointment of Scrutineers:

Motion: "THAT Dr. Elinor F. E. Black and Dr. G. P. Fahrni be appointed scrutineers, and Dr. Murray Campbell and Dr. L. A. Sigurdson be appointed alternate scrutineers." Carried.

7. Reading of Communications, Petitions, etc., to the Council.**A. Request from Faculty of Medicine re Employing Medical Artist.**

The Registrar presented a communication from the Faculty of Medicine advising that an artist completing his final year at the School of Medical Art at the University of Toronto had been interviewed and would be available to start work in Winnipeg on January 1st, 1955. The Dean of the Medical School has assured that space would be made available for a studio in the Medical Buildings, and the usual basic salary for a medical artist is about \$3,600.00 a year, in addition to which the artist can do work on a fee for service basis. The communication requested a grant of money from the College of Physicians and Surgeons of Manitoba to bring the artist to Winnipeg. The Deer Lodge Veterans Hospital are willing to pay one quarter of the basic salary, the Manitoba Institute for the Advancement of Medical Education will also contribute to the salary, some money will be available from the University of Manitoba, and it was hoped that the C.P. & S. might consider making a donation of \$500.00 to \$1,000.00 either to be applied as salary for the first year for the artist, or to help pay for the expense involved in setting up a proper studio.

Motion: "THAT the matter of making a donation re the employing of a medical artist by the University of Manitoba be referred to the Executive Committee, as Council does not feel they can deal with the request at the present time." Carried.

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Department of Health and Public Welfare
Comparisons Communicable Diseases — Manitoba (Whites and Indians)

DISEASES	1955		1954		Total	
	Jan. 30 to Feb. 26, '55	Jan. 2 to Jan. 29, '55	Jan. 24 to Feb. 20, '54	Jan. 1 to Jan. 23, '54	Jan. 2 to Feb. 26, '55	Jan. 1 to Feb. 10, '54
Anterior Poliomyelitis	1	0	8	6	1	14
Chickenpox	203	157	187	192	360	379
Diphtheria	0	1	0	0	1	0
Diarrhoea and Enteritis, under 1 year	6	2	14	8	8	22
Diphtheria Carriers	2	0	0	0	2	0
Dysentery—Amoebic	0	0	0	0	0	0
Dysentery—Bacillary	0	1	3	0	1	3
Erysipelas	2	0	3	1	2	4
Encephalitis	0	0	0	0	0	0
Influenza	2	6	7	4	8	11
Measles	606	251	81	57	857	138
Measles—German	8	4	5	0	12	5
Meningococcal Meningitis	3	2	2	0	5	2
Mumps	214	114	150	62	328	212
Ophthalmia Neonatorum	1	0	0	0	1	0
Puerperal Fever	0	0	0	0	0	0
Scarlet Fever	30	23	70	61	53	131
Septic Sore Throat	6	0	3	5	6	8
Smallpox	0	0	0	0	0	0
Tetanus	0	0	0	0	0	0
Trachoma	0	0	0	0	0	0
Tuberculosis	36	16	29	7	52	36
Typhoid Fever	0	0	2	0	0	2
Typhoid Paratyphoid	0	0	0	0	0	0
Typhoid Carriers	0	0	0	0	0	0
Undulant Fever	0	0	0	0	0	0
Whooping Cough	92	63	5	4	155	9
Gonorrhoea	93	80	95	101	173	196
Syphilis	19	4	8	3	23	11
Jaundice Infectious	45	23	32	14	68	46

Four-week Period January 30th to February 26th, 1955

DEATHS FROM REPORTABLE DISEASES

February, 1955

DISEASES (White Cases Only)	*228,000 Manitoba	*861,000 Saskatchewan	*2,825,000 Ontario	*2,932,000 Minnesota
Anterior Poliomyelitis	1	1	1	3
Chickenpox	203	63	2166	—
Diarrhoea and Enteritis, under 1 yr.	6	16	—	—
Diphtheria	—	—	—	4
Diphtheria Carriers	2	—	—	—
Dysentery—Amoebic	—	—	—	2
Dysentery—Bacillary	—	2	17	5
Encephalitis, Infectious	—	2	—	—
Erysipelas	2	—	7	—
Influenza	2	11	†4716	1
Jaundice, Infectious	45	115	120	177
Measles	606	22	1671	1826
German Measles	8	—	1724	—
Meningitis Meningococcus	3	—	10	8
Mumps	214	3	1874	—
Ophthal. Neonat.	1	—	—	—
Puerperal Fever	—	—	—	—
Scarlet Fever	30	22	294	79
Septic Sore Throat	6	34	2	69
Smallpox	—	—	—	—
Tetanus	—	—	—	—
Trachoma	—	—	—	—
Tuberculosis	36	33	103	64
Tularemia	—	—	1	—
Typhoid Fever	—	1	2	1
Typh. Para-Typhoid	—	—	1	—
Typhoid Carrier	—	—	—	—
Undulant Fever	—	—	3	3
Whooping Cough	92	38	709	76
Gonorrhoea	93	—	182	—
Syphilis	19	—	78	—

†3502 of these cases were in the four-week period ended January 29th, 1955.

Urban—Cancer, 55; Measles, 1; Pneumonia, Lobar (490), 4; Pneumonia (other forms), 8; Poliomyelitis, 1; Tuberculosis, 3; Whooping Cough, 1. Other deaths under 1 year, 7. Other deaths over 1 year, 177. Stillbirths, 17. Total, 274.

Rural—Cancer, 33; Pneumonia, Lobar (490), 2; Pneumonia (other forms), 7; Syphilis, 2; Tuberculosis, 5; Diarrhoea and Enteritis, 2. Other deaths under 1 year, 11. Other deaths over 1 year, 189. Stillbirths, 10. Total, 261.

Indians — Pneumonia (other forms), 6; Chickenpox, 1; Diarrhoea and Enteritis, 4. Other deaths under 1 year, 2. Other deaths over 1 year, 4. Stillbirths, 18. Total, 18. †White death on Indian Reserve.

Anterior Poliomyelitis—One case reported to date this year. This was a girl with slight paralysis, residence in St. Vital.

Vaccination Against Poliomyelitis—Although the report of the Field Trial carried out in 1954 will not be available until early April we are busy planning for the vaccination of six and seven year olds throughout the province whose parents wish them to have the vaccine.

Diphtheria—No more cases have been reported in Manitoba and the one case and two carriers now show negative swabs. The source of the infection has not yet been discovered.

Measles are quite prevalent and giving the province a "spotty" look.

Influenza—Although not appearing to any extent in our report, we know from hearsay to be quite prevalent in the province. One institution has had about a hundred and fifty cases to date. It has not been necessary to send any of these to hospital but some have been moderately ill.

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Book Reviews

Antibiotics and Antibiotic Therapy, by Allen E. Hussar, M.D. and Howard L. Holley, M.D., pp. 475. Price \$6.00. The MacMillan Company, New York, 1954.

This book fills a long felt need for a clinical manual on antibiotic therapy. The latter, representing, as it does, the greatest therapeutic advance of the century, has been surprisingly relegated to medical journals, monographs, and publications of drug houses. Now, with the publication of this authoritative and comprehensive primer, the student and the clinician will find at their disposal a quick reference and a sorely needed guide.

The book is divided into three parts. The first part deals with fundamental principles, the second with properties of individual drugs, and the third, under the heading "The Drug of Choice", gives their specific indications. Well arranged, well indexed, and replete with references, this book will be welcomed by everyone interested in this subject.

S. V.

Handbook of Treatment, by Harold Thomas Hyman, M.D., pp. 511. Price \$6.50. J. B. Lippincott Company, Philadelphia and Montreal, 1955.

This handbook differs from others, bearing the same or similar title, in that it can be read without the aid of a magnifying glass, for its print is large enough to be legible with the naked eye. Moreover, it is more presentable, for it is well bound, a feature which, while detracting somewhat from its portability (one would hesitate to throw it carelessly into an untidy instrument bag) greatly enhances its appearance. It is also well arranged and indexed—3900 entries.

Treatment is discussed under four headings: (1) immediate care, (2) continuous care (favorable course), (3) continuous care (unfavorable course), (4) continuous care (progressively unfavorable course). A discourse on general principles, which includes etiology, pathology, physiology and classification, as well as advice on diagnostic procedures to be adopted in various situations, precede and accompany the therapeutic instructions.

One cannot help feeling that this attempt at all-inclusiveness is a source of weakness rather than strength, an invitation to errors, misnomers and dogmatic assertions which are often unwarranted. As examples of these may be quoted the following passages: "Coagulation acceleration may be accomplished by systemically administered heparins" (page 41), or: "If the decompensated patient's rhythm is of sinus origin, try first to accomplish diuresis with a subcutaneous or an intramuscular injection of a mercurial, as above; and reserve digitalization for supplementation of the diuretic, if needed." (page 78).

As long as the reader is aware of these shortcomings, and keeps his critical sense alert, he will find the book useful. He will, particularly, appreciate the rosters of drugs which are exhaustive and up to date.

S. V.

Hormones, Health, and Happiness. Warren H. Orr, M.D. The MacMillan Co., New York, 1954, 322 pp. Price \$4.50.

This is a simple, nontechnical book, written primarily for laymen. The author feels that it is important for the general public to have a knowledge of endocrinology, "so that glandular problems can be readily observed by parents and associates." This will allow "untold thousands" to be treated "early enough to be saved for more productive lives". The author hopes there are many symptoms of dysfunction of the various endocrines that the layman will be able to recognize, after he has read the book. This reading, however, should not replace a "periodic endocrine checkup", which should be a part of the physical checkup.

The book is divided into three parts. Part I is entitled, "How Glands and Hormones Affect You". Part II is called "Special Problems". Part III is entitled, "Glands and Hormones". There is a glossary at the end of the book, and a formidable list of recommended books for further reading.

Part III is the best part of the book, and might have been placed first. Throughout, there are many case reports showing how endocrine dysfunction can adversely affect patients of all age groups. The average layman, however, would be unable to recognize early endocrine imbalance after reading this book, and would certainly mistake many other conditions for endocrine disease. Nor would the average physician fare much better, for there are many examples in which the author's diagnosis and medical interpretations could be questioned. On the whole, however, the book is readable, and interesting.

A. R.

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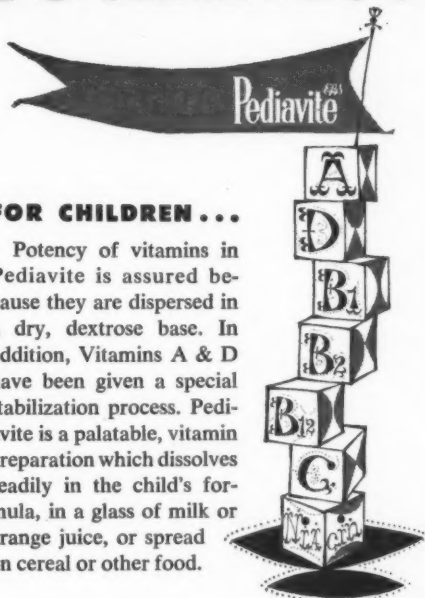
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